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# BRAIN ABSCESS
## ITS SURGICAL PATHOLOGY
AND
## OPERATIVE TECHNIC

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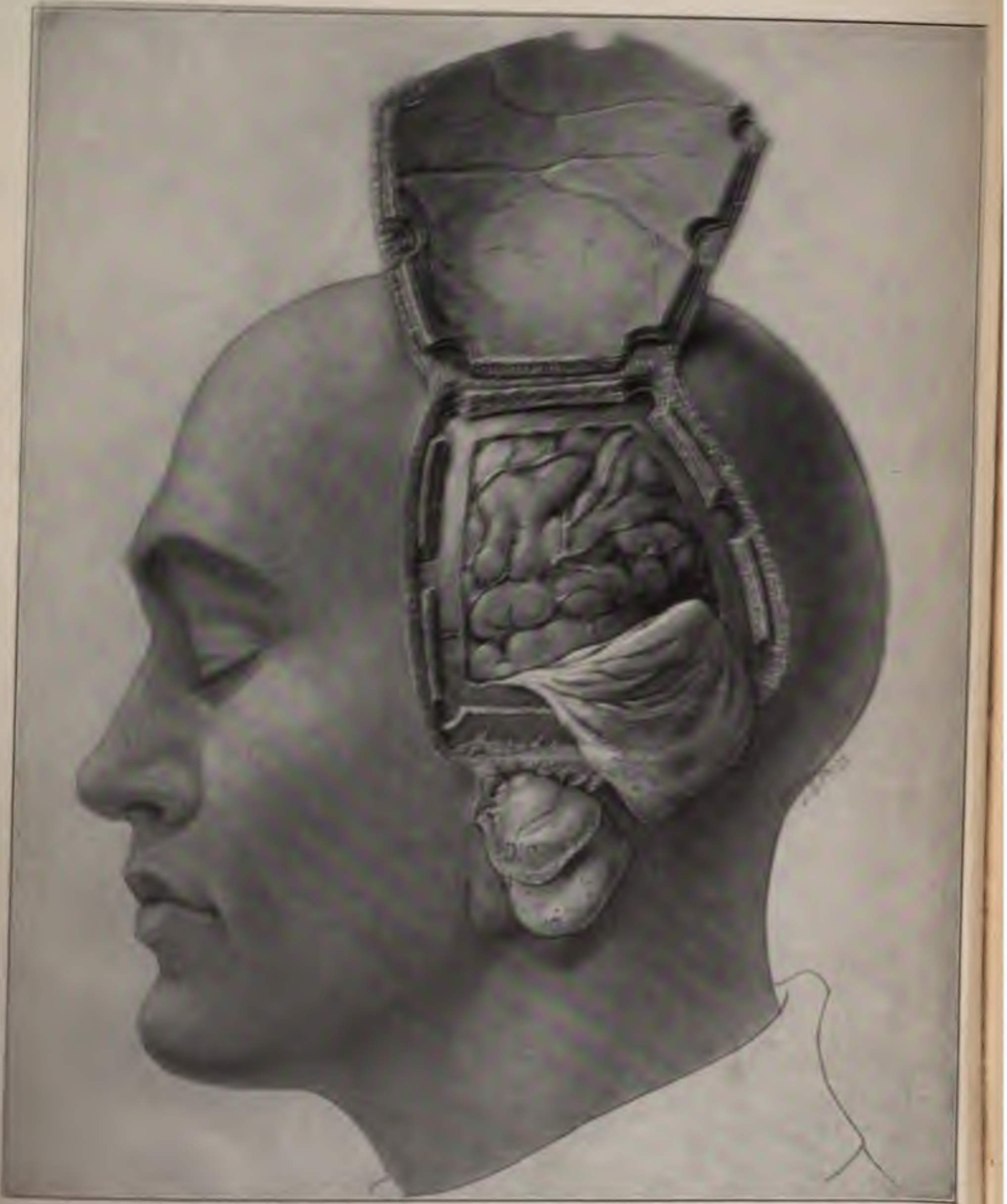

FIG. 1.—Frontispiece. The Osteoplastic Flap for Exploration for Abscess of Middle Fossa.

# BRAIN ABSCESS

## *ITS SURGICAL PATHOLOGY*
## *AND OPERATIVE TECHNIC*

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1922

To the
Memory of My Father
who taught me "If all were frankly to record
their thoughts and experiences, even
of one day, humanity would be
advanced centuries," and
TO MY WIFE
the inspiration of the book.

53645

# FOREWORD

This monograph is the result of considerable intradural experience, associated with many bitter disappointments and failures; some animal experimentation; and of observations during post-mortem examination of cases in which death had resulted from varied intracranial lesions. Recognizing that surgical as well as pathological and diagnostic information is obtainable from post-mortems, a personal effort invariably has been made to obtain an autopsy on all cerebral cases coming under my observation. The fatal cases previously examined or operated upon, on which post-mortems were not performed, are very few.

The laity are anxious to assist the surgeon in obtaining whatever knowledge may be derived from an examination of their dead, provided they feel that the surgeon has striven earnestly to assist them, and that the post-mortem will be reverently conducted.

Cushing, whose genius has transformed non-suppurative intracranial surgery into comparatively safe, delicate, almost bloodless physiological procedures, has long contended that the ordinary surgical technic applicable to other parts of the body is insufficient to combat successfully the unique physiological factors and pathological changes presented within the dura; and it is Cushing's technic which the author largely has used in operating for intracranial suppuration.

Surgically considered, intracerebral abscess stands in a class by itself, the problems presented in its surgical treatment differing from those of suppuration in other parts of the body and distinct from those encountered in the treatment of non-suppurative lesions of the brain. As many of the technical problems presented still remain far from solution, it is proposed to call attention to some of these physiological factors, their alteration by pathological processes, and the technic which, to the author's mind, most satisfactorily meets them. The success of intradural surgery always will depend upon utilizing every advantage offered by the preventive and reactive efforts of nature, while a single technical error may mean the loss of the patient's life or at least of his subsequent usefulness.

In this monograph the writer has attempted to correlate the surgical treatment of the different pathological lesions of brain abscess, for he believes that, although pathology and treatment usually are dealt with in separate chapters, they are in reality one and should be so considered. Certain it is that if one is to meet with even a moderate degree of success in the treatment of brain abscess, the surgical manipulation in each individual case must be adapted to the exact pathological condition present.

With this object in view emphasis is laid upon the pathology and on mechanical changes, and a classification is adopted which is capable of individual clinical recognition, because the author believes that our present knowledge should enable us not only confidently to diagnose the presence of brain abscess, but also in a large proportion of the cases to determine its location and whether or not it is surrounded by a capsule.

To accomplish this the surgeon must divest himself of certain ideas inherent in the old classification of aural and nasal origins, and must conceive of them not according to their immediate origin, but pathologically, according to the causative lesion of entrance into the central nervous system; that is, whether extension is by direct tissue suppuration or by retrograde thrombophlebitis, for which the author suggests the terms Adjacent (Secondary), and Intercurrent (Tertiary), when immediately occasioned by a secondary gross lesion such as sinus thrombosis, to differentiate both from abscesses originating from the deposit of infected bacterial virus circulating free in the blood stream, the latter being pathologically metastatic, even though the original focus of infection may be in the ear or the nose. One of the chief objects of this work is to enable the surgeon to apply a pathological nomenclature to the subject and thus approach the treatment of brain abscess in a pathological as well as a technical mental attitude.

No apologies are offered for the personal character of the book. It is largely an account of experience in more than fifty cases of adjacent brain abscess which have come under the author's observation. It has been written because he recognizes that many deaths might have been avoided had he earlier been in possession of the knowledge he has gained by his failures. He has analyzed his cases, making an effort in each particular case to learn why the patient recovered or ied.

In the chapter on Diagnosis the writer voices many opinions ich must be regarded as purely speculative, much of which doubt- will be demonstrated to be fallacious as our physico-pathological understanding of the clinical manifestations of suppurative cerebral lesions increases. For this he offers no excuse, believing that a better understanding of disease often may be obtained by speculation, provided the speculation is founded on the available clinical and experimental evidence.

Nor does he apologize for the minutely detailed technic. He has emphasized the technic, rather, because he has learned from personal experience to regard it as equally important with diagnosis. While the opinions expressed are his own, he has endeavored whenever possible to substantiate them by reference to recorded opinions and observations. When an unsubstantiated opinion is cited, the authority for it is cited in the text.

In the preparation of the book the author's thanks are due to Dr. Frederick A. Sutton and Dr. Harrison S. Martland for the pathological examinations and sections; to the staff of the Newark Eye and Ear Infirmary for placing at the author's disposal the records of cases under their care; to Dr. F. Robbins for compilation of the cases of frontal and cerebellar abscess and for other valuable cooperation; to Mr. Edward B. Mead for helpful mechanical aid in the construction of instruments, and to the author's secretary, Miss Catherine Fideles Casey, for indispensable assistance in the preparation of the manuscript.

WELLS P. EAGLETON.

Newark, N. J., January 2, 1922.

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# PART I.

# GENERAL CONSIDERATIONS IN INTRACRANIAL SURGERY.

# CHAPTER I.

# PREPARATION.

### MENTAL ATTITUDE OF THE SURGEON.

In approaching the problems involved in localized, intradural suppuration the surgeon must cultivate a peculiar mental attitude which will render him sensitive and attentive to the minute details of diagnosis and of surgical procedure. In no other branch of surgery may the advisability of a major operation depend upon such apparently trivial manifestations; in no other part of the body will neglect of slight hemorrhage play so important a part in the recovery or in the future well-being of the patient; in no other branch of surgery is preparedness for operation more important or team work more essential. The surgeon who postpones an operation for brain abscess to suit his personal convenience starts with a handicap that too often results in the death of his patient—a death frequently avoidable.

### IMPORTANCE OF PROPER HISTORY TAKING BEFORE EXPLORATION.

Many patients suffering from brain abscess are brought to the hospital in a dazed condition, unable to furnish a reliable account of their illness. The possibility of a brain abscess, then, may rest upon the presence of a purulent discharge from the ear or nose, a bronchiectasis, or the scar of a previous head injury, and the diagnosis or elimination of such abscess can only be made by the expenditure of time and patience in interviewing persons who are in possession of accurate information about the patient prior to the appearance of pronounced cerebral symptoms. This has been accomplished many times, and has resulted in the greatest personal satisfaction.

The following apparently trivial, yet really important, diagnostic details have thus been ascertained:—The presence and exact date of the initial "vague chill," from which could be calculated the probable duration of the abscess, and whether it was acute without limiting membrane, or chronic with limiting membrane; the occurrence of attacks of uncontrollable vertigo, knowledge of which has largely influenced the selection of the cerebellum as the site for exploration; transient periods of a "dreamy" state; transient or permanent aphasia; complete motor, sensory, or "naming" ataxia; the gradual or sudden onset of hemiplegia—the involvement of the hand, leg and face being separated by appreciable intervals; and the presence of an unrecognized hemianopsia. Some or all of these symptoms always occur in the development of an abscess of the brain, and a knowledge of their presence is of incalculable value to the surgeon in making his diagnosis.

### HISTORY TAKING AND COMPLETE NEUROLOGICAL EXAMINATION PRIOR TO APPEARANCE OF SEVERE CEREBRAL SYMPTOMS.

It is the practice among many surgeons when treating suppurative diseases of the ear or nose associated with slight but suspicious symptoms—vague chill, headache, dizziness, slight subnormal temperature, slow pulse—to await the appearance of outspoken cerebral symptoms before making a complete neurological examination. (See Appendix I, "Guide for Neurological Examination.") This frequently results in the performance of an intracranial operation without that minute and accurate information, previously obtainable, upon which depends the adoption of the correct exploratory procedure.

Examination of the recorded cases [1] of brain abscess shows that a very large proportion had been operated upon previously for mastoid disease, accessory sinus suppuration, or compound fracture of the skull. The radical mastoid operation has frequently been performed for symptoms—headache and dizziness—the intracranial origin of which was not apparent until some time after the operation.

In the presence of a wound or scar from a suppurative lesion of a cranial bone, or any suppurative disease capable of causing metastatic brain abscess, a chill, no matter how vague, followed by a slightly subnormal temperature, demands not only a complete neurological examination, but repeated blood cultures; while slight cerebral symptoms, such as persistent headache, attacks of dizziness, slow pulse, subnormal temperature and slight psychic changes, call for the removal of the patient to a hospital and the performance of a lumbar puncture, repeated if necessary until a positive diagnosis is obtained.

The association of brain abscess and sinus thrombosis demands the early recognition of the blood-stream contamination in order to differentiate between the symptoms due to sinus thrombosis and to intracerebral suppuration. On more than one occasion, in the presence of a papillœdema secondary to occlusion of the venous return circulation by a thrombus, one positive blood culture was the deciding factor against intradural exploration. On the other hand, in a number of instances the operative finding of an occluding sinus thrombosis has caused the author to overlook an abscess of the cerebellum. In one case, at least, the thrombus was undoubtedly aseptic and secondary to the abscess. Repeated negative blood cultures during the period of observation would have indicated exploration of the brain.

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[1] See Appendix II and Appendix III, recorded cases of cerebellar and frontal lobe abscess.

The diagnosis of a secondary "protective" (aseptic) meningitis [2, 3, 4, 5, 6]—a frequent manifestation of the effort of the intracranial contents to wall off the suppuration—is of the greatest value in the early recognition of the localized intradural suppuration, while the early diagnosis of a general streptococcic and pneumococcic meningitis, both of which may exist for some time without producing marked symptoms, has frequently prevented a useless exploration.

**Time for Operation.**—Brain abscess, once the diagnosis has been made, permits of no delay or temporizing; it demands the immediate sacrifice of all other interests. Two cases illustrate the importance of prompt surgical intervention.

CASE I, R. F.

Cerebellar Abscess. Male, age 34; running ear for fifteen years. Seen at eleven A. M. on the tenth day of illness. Temperature 100°; pulse 54; severe dizziness; intense pain in occiput; occasional vomiting; double optic neuritis. Had been delirious previous night. No tenderness over mastoid; no discharge from ear.

**Diagnosis.**—Cerebellar abscess. Ordered to hospital and preparations made for operation. Patient did not report as ordered. Late in the day he arrived in good condition; walked up two flights of stairs, needing only the help of the balustrade because of dizziness. Operation appointed for next day. Less than four hours after admission the patient suddenly became maniacal, had a convulsion, and expired within a few minutes. The abscess had ruptured.

CASE II, C. M.

Cerebral Abscess. Girl; chronic discharging ear; headache for several weeks; vague chill; attacks of dreamy state; papillœdema; paresis of external ocular muscle; heminapsia. When examined in excellent condition, rational and strong.

**Diagnosis.**—Cerebral abscess. Operation advised. Attending physician unwilling to consent without permission of family, who did not regard girl as seriously ill. Delay in obtaining consent. Next morning physician reported that patient was so much better there must be doubt of brain abscess, but family

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[2] "Meningitis Aseptica": Reischig; *Zeit. f. Ohrenheilkunde*, 1913, Bd. 69, p. 78.

[3] "Meningitis Sympathetica": Plaut und Schottmüller; *Leitfaden zur Untersuchung der Cerebrospinalflüssigkeit*.

[4] "Meningitis Sympathetica": Strauss, Israel; *Amer. Jour. Med. Soc.*, November, 1917, Vol. CLIV, No. 5, p. 748.

[5] "Protective Meningitis": Martland, Harrison S., Personal Communication.

[6] Case XVII, M. D., Metastatic Abscess, Chapter Five, p. 77.

**consented to operation. Afternoon appointed for operation. Suddenly severe rigor and convulsions and patient passed into coma. Strong pulse but respiratory paralysis. Artificial respiration failed to sustain pulse while hasty preparations for operation were being made.**

**Autopsy.—Large temporo-occipital lobe abscess with capsule which could have been drained by proposed operation with an excellent chance of recovery.**

Four other fatal cases have come to the author's attention in which the surgeon allowed external circumstances to occasion a postponement of the operation after the diagnosis of brain abscess had been made. In these four cases one abscess was temporo-sphenoidal, one occipital, one frontal and one cerebellar.

While it is well recognized that sudden death results from rupture of a cerebellar abscess, it is not sufficiently appreciated that abscesses in other parts of the brain frequently cause death without warning.

**Assistants.**—In no branch of surgery is trained team work as necessary as in intradural surgery. In many hospitals the operator is assisted by a house surgeon and a nurse and the anesthetic is administered by a junior house surgeon—a system which frequently results in the neglect of small details on which the success of an intradural operation essentially depends. To be prepared to meet the technical difficulties which may be encountered during any intradural operation, a team of not less than four persons trained in brain surgery is required—operator, assistant, anesthetist and chief nurse. Furthermore, it is desirable to have two assistants skilled in applying artificial respiration and direct blood transfusion. The first assistant and the chief nurse should have an understanding of the operator's technic, inasmuch as the assistant must anticipate every surgical need. To allow an assistant imperfectly trained in intradural surgery to participate actively in the operative work is to invite disaster. I have more than once "lost" an abscess before its complete evacuation because the assistant was not sufficiently trained to respond quickly to the emergency. Likewise, I have had an assistant well trained in general surgery but without intradural experience, and consequently not appreciative of the extreme delicacy of the wall of a non-capsulated brain abscess, cause the searching canula which had been placed within the abscess cavity to perforate the delicate wall between the abscess and the adjacent lateral ventricle.

**Anesthesia.**—Ether is the anesthetic of choice except when the patient is in such deep coma that no anesthetic is required. The frequency of embarrassed respiration with its associated increased bleeding demands that all intradural operations be conducted under anesthesia,[7] administered by an expert with an apparatus capable of exact dosage. *Horsley*[8] used a mixture of chloroform and oxygen. The author has not adopted this as it does not appear to him that the hemorrhage would be lessened by this method though theoretically it should be advantageous. *Cushing*[9] advocates a local anesthetic for cranial war injuries and experience with local anesthesia in the highly sensitive area of infected mastoids convinces the author that many cases of brain abscess may be explored under local anesthesia.

**Technic.**—The success of local anesthesia in cranial bone surgery depends upon: (1) Systematic infiltration; (2) avoidance of pulling by instruments—retractors, artery clamps, etc.; (3) avoidance of all transmitted shocks—chiseling or hammering; (4) performance of all bone work by cutting or biting instruments such as electric or hand-driven burrs, rongeurs, gauges, Gigli saws, etc. The dura is not a sensitive structure; it requires only the application of a local anesthetic. The author has frequently slit the dura even without a local anesthetic. The brain tissue itself is non-sensitive.

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[7] Heuer and Dandy: "A Report of Seventy Cases of Brain Tumor"; *Johns Hopkins Hospital Bulletin*, August, 1916, p. 333.

[8] Horsley, V.: "On the Technic of Operations on the Central Nervous System"; *Brit. Med. Jour.*, 1906, II, p. 411.

[9] Cushing, H.: "Concerning Operations for the Craniocerebral Wounds of Modern Warfare"; *Military Surgeon*, 1916, Vol. 38, p. 601.

# CHAPTER II.

# GENERAL SURGICAL TECHNIC OF INTRACRANIAL OPERATIONS.

## GENERAL SURGICAL TECHNIC.

**Position of Patient.**—In all intradural explorations the field mapped out by the original incision should be horizontal with the operating table and provision should be made for its maintenance without muscular strain on the patient.

**Technic.**—The table should be flat and covered by a thick mattress with a rubber casing. A sloping table renders impossible the proper position of the head. The neck allows a certain degree of latitude by which the head can be rotated from the perpendicular of the body. This position can be maintained by the patient without strain or interference with respiration. The extent of the rotation varies with the individual patient and can be determined only by placing the patient on the table before administering the anesthetic, finding the degree of rotation available, and then adapting the supporting and restraining apparatus to the individual requirements.

In a temporo-sphenoidal lobe exploration the patient's body lies on the table with the head rotated sufficiently for the occipito-bregmatic line to be exactly horizontal with the plane of the table. The position is maintained by padding with sandbags under the head and neck, thus elevating the shoulders, back and buttocks. While the patient is under the anesthetic occasional slight pressure of the hand of the anesthetist applied to the jaw will prevent the backward rotation of the patient's head. In frontal lobe exploration the patient lies prone. In cerebellar abscess, if the exploration is to be behind the sinus, the patient is supported by his abdomen and shoulders with chest and face pointing directly downward, an "outrigger" being used. If the exploration is to be in the anterior portion of the cerebellum one shoulder of the patient is elevated more than the other and the head slightly rotated so that the face points downward forty-five degrees from the vertical—Sims' chest position.

**Utilization of Gravity.**—A device permitting the rapid conversion of the operative field to admit of complete evacuation of a brain abscess by gravity is of material assistance. In one case of encapsulated intrapiarachnoid abscess, after the evacuation of as much pus as would flow, the patient, a small child, was elevated by the feet and the operated side turned downward. This was followed by the escape of an additional large quantity of pus.

Failure to utilize gravity has resulted in numerous instances in incomplete evacuation, the abscess having been punctured at its uppermost extremity. In such a condition the elevation of the foot of the table causes a recurrence of the flow of pus after its cessation—always desirable and frequently the deciding factor for success. For complete primary evacuation the table should be equipped with a device to allow of depression and elevation of the head and rotation of the patient to one side or the other.

**Head-rests.**—The author has tried head-rests attachable to a general operating table but they have not proved thoroughly satisfactory. F. Krause, of Berlin, while performing an occipital operation, had the patient in a sitting position, the head inclined forward, resting against the chest of an attendant who steadied the patient's head between his hands.

For cerebellar operations the "outrigger," with shoulder supports to allow of free respiration, is most satisfactory.[1, 2, 3]

**Elevation of the Head.**—Elevation of the patient's head during the operation lessens hemorrhage. Some surgeons operate with the head and shoulders elevated at an angle of about thirty degrees. This, however, necessitates a constrained position for the operator which, especially in a "temperamental" man, seriously interferes with his efficiency, particularly toward the end of a long operation.

**Technic.**—The operating table should be at least twelve inches higher than the standard tables used for abdominal operations. The operator and assistants should stand squarely on their feet, without strain, thus enabling them to work for several hours without unnecessary fatigue. Each should attain the height at which he works best, by an individual platform.

LIMITATION OF THE FIELD:

**Technic.**—After marking the proposed incision with a knife, the head is covered with gauze, the operative field being left exposed and surrounded by toweling pinned to the scalp, a wall of toweling being erected between the anesthetist and the operator so that it is impossible for the anesthetist's hand to invade the operative field or for the operator to go beyond it.[4] By pinning the toweling to the skin with delicately constructed clips an area with a margin of one-half inch may be walled off.

**Control of Hemorrhage.**—One of the imperfectly solved problems of cranial surgery is the control of hemorrhage. The number of operations which result fatally, directly or indirectly from bleeding, is still large. Many surgeons, chiefly because of failure to control hemorrhage, divide all intradural operations associated with marked increase of the intracranial pressure into two stages—at one

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[1] Frazier, C. H.: "Problems and Procedures in Cranial Surgery"; *Jour. Am. Med. Assoc.*, June 5, 1909, Vol. 52, p. 1805.

[2] Smith, H. B.: "An Apparatus for Supporting and Holding the Head and Shoulders in Cerebellar and High Spinal Operations"; *Jour. Am. Med. Assoc.*, Nov. 26, 1910, Vol. 55, p. 1859.

[3] Cushing, H.: "Tumors of the Nervus Acusticus"; 1917, *W. B. Saunders Co.*, Phila. and London, p. 252.

[4] Cushing, Harvey: "Technical Methods of Performing Certain Cranial Operations"; *Surg. Gyn., and Obs.*, 1908, March, Vol. 6, No. 3, p. 227.

operation turning down the osteoplastic flap, and at a later time opening the dura. With proper attention to hemostasis this should rarely be necessary.

*Hemorrhage from the Scalp.*—In cases with greatly increased intracranial pressure bleeding from the scalp may be very severe. In addition to the many spurting points there is a general oozing which, though temporarily of little moment, may seriously militate against the patient's recovery if allowed to continue. Over both cerebrum and cerebellum the hemorrhage can be controlled by firm manual pressure against the skull and the subsequent reflection of the galea over the cut surface.

The excessive general oozing so frequent during operations for brain tumor does not occur during exploration for cerebral or cerebellar abscess, because in the presence of intradural suppuration, long before the development of the excessive increase of intracranial pressure so frequently associated with tumors, the patient has expired. Continued excessive increase of intracranial pressure is not compatible with life in the presence of suppuration.

**Technic of Controlling Hemorrhage.**—The assistant, with the finger tips of both hands, presses the scalp firmly against the bone while the surgeon cuts in the small area between them, checking the hemorrhage by catching the galea with fine artery forceps and reflecting it over the cut surface. The artery forceps controls the hemorrhage, not by actual compression, but by pulling the galea over the vessel.

The assistant must not remove the pressure until the hemorrhage is completely controlled. The number of forceps necessary is very large. The clamps are dropped into the open palm of an assistant, for if allowed to hang they will become detached by their own weight or during manipulation. The assistant keeps the forceps absolutely flat and in proper relation to each other. Straps of gauze wrapped around or through the handles of bundles of ten or twelve forceps allow the assistant to control a large number. After the application of the forceps a band of flat gauze pulled tightly over the cut surface controls the remaining ooze and protects the forceps from displacement. Sufficient attention to this simple method prevents loss of blood and shortens the time of operation.

The author has attempted ligation of the clamped vessels before closure; but it has been found to consume time and is of very little value.

During closure, with the exception of actual spurters, the hemorrhage must be controlled by exact and careful approximation of the cut surfaces by suturing, layer by layer, no dead spaces whatever being allowed to remain. Because of the adhesion of the galea to the pericranium and subcutaneous tissue the soft parts must be pushed, not pulled, together by an assistant, all traction by sutures being avoided. The process is long and tiresome, but it is the only one that is uniformly attended with success.

*Tourniquets and Compressors.*—When applicable in abscess well above the floor, metastatic or following trauma, a rubber tourniquet[5] materially lessens the bleeding. On several occasions, however, the tourniquet interfered with the proper exposure of the abscess which, being subdural, had gravitated downward. Removal of the tourniquet with an open dura is associated with annoying bleeding which may be difficult to control.

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[5] Cushing, Harvey: "Pneumatic Tourniquets, with Special Reference to their Use in Craniotomies"; *Medical News*, March 26, 1904, Vol. 84, p. 577.

Heuer's temporo-muscular clamp, a modification of an intestinal clamp, materially lessens the bleeding but in temporo-sphenoidal abscess, as the exposure must be low down, it cannot be employed.

The spring clips of Makkas are distinctly useful when respiratory or circulatory troubles necessitates very rapid exposure. They slip, however, if applied near the edge of the wound rendering closure difficult. They also severely bruise the tissue, frequently causing local necrosis.

Heidenhain's method of using deep sutures of heavy silk or silkworm gut passed with a large, full curved needle through skin to bone, one overlapping the other, does not control the hemorrhage but seriously interferes with the closure and is followed by stitch abscesses.

Kredel's plates, likewise, have similarly failed.

### PERFORATION OF SKULL AND FORMATION OF OSTEOPLASTIC FLAP:

**Instruments.**—Hudson's perforators save time, but it requires a little experience to recognize correctly the sensation when the skull is perforated; otherwise it is possible to injure the dura and damage the brain substance with them. *Hartley* and *Kenyon's* perforator,[6] driven by an electric motor, is somewhat dangerous, but properly used it makes possible a rapid and safe perforation of the skull. *Frazier's* electric fraise[7] neither works rapidly nor allows a proper bevel.

### SACRIFICE OF BONE:

**Instruments.**—When a large area of bone is to be sacrificed, as in cerebellar abscess, the usual gauges and chisels are too small. The author is using more and more very large chisels, rongeurs and heavy hammers. A large chisel properly placed is not so dangerous an instrument as a small one, but it requires a heavier hammer to drive it. Herman Bleyle has made a well-balanced hammer weighing nearly twelve ounces; but the author has not yet found the rongeur strong enough and powerful enough to cut bone as rapidly as is necessary. The French bulldog rongeurs and the long-handled rongeurs devised by Bryant[8] are the most satisfactory.

## CONTROL OF MODERATE BLEEDING FROM THE DIPLOIC VESSELS DURING THE PERFORATION OF THE SKULL.

Disregard of moderate bleeding results in unnecessary loss of blood. Moderate hemorrhage can be controlled by cotton or wax until the dural opening is enlarged, when the venous hemorrhage generally ceases entirely.

**Technic.**—Horsley's wax must be of the proper consistency. There is a little knack required for its successful application. It must be WIPED, not pressed, into the bony foramen. The proper pressure to be exerted comes with experience. Attempts to control hemorrhage by all forms of stiff plugs—Krause's hooks, ivory or wooden plugs—are useless as they are constructed on a wrong principle, namely, that the bleeding has to be overcome by force whereas in reality it requires complete occlusion of the vessels. The slightest pressure completely stops bleeding. A frequent cause of failure is the attempt to wipe wax into a vessel which is still partially outside of the bony foramen.

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[6] Hartley and Kenyon: Experiences in Cerebral Surgery; *Annals of Surgery,* April, 1907, Vol. 45, p. 481.

[7] Frazier, C. H.: Problems and Procedures in Cranial Surgery; *Journal American Medical Association,* June 5, 1909, Vol. 52, p. 1805.

[8] Bryant, William Sohier: An Improved Rongeur for Mastoid Operations; *Journal American Medical Association,* August 6, 1910, Vol. 55, p. 502.

### BULGING OF THE DURA INTO THE PERFORATION OPENINGS:

If the intracranial pressure is greatly increased on making the initial opening the dura bulges forcibly into the opening and it is with difficulty that a director can be passed from one to the other. The proper use of the dural separators is acquired only after a little experience. A large number of dural separators of different sizes and shapes is required. The instrument should be advanced carefully, by gentle pressure and a to-and-fro movement. The introduction of the protected guide for the Gigli saw is facilitated by bending its tip. The bulging of the dura through the opening can be prevented by the performance of a lumbar puncture. This is frequently attended with severe hemorrhage, which can be completely controlled only after the bone flap has been elevated.

### TECHNIC OF VENTRICULAR PUNCTURE:

A trephine opening is made in the occipital bone about three-quarters of an inch above the transverse sinus and three-fifths of an inch from the median line; the dural opening need only be large enough to admit the searcher. The searcher is passed directly forward; if two or three punctures fail to reach the ventricle the opposite hemisphere should be similarly trephined and punctured, as obliteration of one lateral ventricle from pressure is a frequent occurrence.

### THE CUTTING OF AN OSTEOPLASTIC FLAP OF THE SKULL:

**Instruments.**—The spaces between the openings are cut with a Gigli saw or with a narrow Marentz rongeur.

**Technic.**—The saw should be held tense, with the handles wide apart so that the cutting angle of the saw will be as obtuse as possible. The movements of the saw should be transmitted partially by rotation of the operator's body rather than by a to-and-fro movement of his arms alone. If the arms alone are used the operator is apt to allow his hands to approach each other, thus breaking the saw. Properly used a Gigli saw will cut through any skull without difficulty, making a bevel upon which the flap can rest. It is the best and safest instrument at our command. (See Part II, p. 41.)

### Control of Hemorrhage from Interior of Skull Before Dura is Opened.

After the elevation of the flap, or sacrifice of the bone, considerable blood occasionally comes from beneath the edge of the bone. The crowding into the bleeding area of a very small roll of cotton with a black thread attached generally controls the bleeding. The hemorrhage is frequently quite profuse at first, but later the small compress can be slowly removed, generally without recurrence of the hemorrhage.

In hemorrhage from a venous sinus the accurate placing of a piece of fascia lata over the rent—the postage-stamp method—will control the bleeding. Its proper application, its unfolding and tucking away with forceps, requires considerable experience.

### CONTROL OF HEMORRHAGE FROM THE DURA ITSELF.

In cases of increased intracranial pressure there is a constant oozing from the small vessels of the dura which, while of slight importance before opening the dura, is extremely annoying later, and is most difficult to control.

Oozing is readily stopped by placing over the oozing points strands of dry cotton, in the meshes of which the blood will coagulate. Upon the removal of the cotton, however, at the end of the operation, the oozing is apt to recur with increased persistence. A properly applied piece of muscle is very effective. It should be placed flat over the bleeding point and the blood pressed from under it. The muscle acts by occlusion and as a chemical hemostatic. Applied blood clot also at times will control a very troublesome oozing. When obtainable, the application of fibrin paper is of the greatest advantage.[9, 10]

The larger vessels of the dura are encircled with a needle and ligated in two places, or "double-clipped," before incision. In ligation by a needle-passed suture I have frequently injured a pial vessel, with resultant annoying hemorrhage.

### CONTROL OF HEMORRHAGE FROM THE PIAL VESSELS.

All hemorrhage must be controlled before the closure of the dura, for if a single point is left bleeding at the primary closure it will continue to ooze, with resulting degeneration, the pressure from the brain itself being insufficient to stop the hemorrhage. The pial vessels—endothelial tubes—run in the substance of the piarachnoid, which itself hugs the cortex, dipping down into each convolution. The pial vessels anastomose freely with each other, and furnish the superficial vessels of the cortex. They cannot therefore be caught and elevated without tearing the adjacent piarachnoid and injuring the superficial cortical vessels; they must be encircled without elevation or laterally occluded by pressure.

**Technic.**—The application of the finest hemostat only increases the bleeding, since it is impossible to elevate the piarachnoid or pial vessels sufficiently to ligate by the forceps. The pial vessels may be ligated by passing a needle ligature around them, but passing the needle and tying the ligature without tearing the tissue is a most delicate procedure. Cushing "double-clips" all pial vessels and cuts between the clips. Horsley double-ligated all pial vessels before cutting. Hartley stopped bleeding by applying with a slight pressure a thick layer of

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[9] Harvey, S. C.: Fibrin Paper as an Hemostatic Agent; *Annals of Surgery*, 1918, Vol. 68, p. 66.

[10] Cushing, Harvey: The Control of Bleeding in Operations for Brain Tumors; *Annals of Surgery*, 1911, Vol. 54, p. 1.

vaseline spread on a piece of gauze. If the gauze is raised the vaseline adheres to the bleeding points and seals them. The use of adrenalin, also, will stop pial bleeding, but the hemorrhage will recur later from the relaxation of the constriction unless pressure is applied.

### Control of Hemorrhage from the Brain Substance.

This is generally slight and is stopped spontaneously by the surrounding tissue. If not, a piece of fascia, or, even better, of temporal muscle, or the fibrin paper plates of Harvey,[11] must be smoothly applied to the bleeding surface. The immediate control of hemorrhage by the smooth application of autogenous tissue over the bleeding part is one of the most beautiful and delicate procedures in surgery. It is rarely necessary in operation for brain abscess to destroy the continuity of the brain substance, for, unlike brain tumors, which must be removed from the place where they are embedded in the brain, an abscess is drained from within without disturbing the surrounding tissues.

### CLOSURE OF DURA:

**Technic.**—Approximation by catgut suture is most unsatisfactory, but in the presence of suppuration the author uses it. Waxed fine silk gives a much better approximation of the dural edges.

In increased intracranial pressure it may be impossible to close the dura without injury to the brain substance. Suturing a transplant of fascia lata into the dural defect makes an excellent cerebral covering.

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Rahm, Hans: Die Mechanik der Gehirnerschütterung; *Centralblatt für Chirurgie*, February 14, 1920, No. 7, p. 146.

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[11] *Loc. cit.*

# PART II.

# SURGICAL PATHOLOGY AND OPERATIVE TECHNIC OF BRAIN ABSCESS.

# CHAPTER III.

# PATHOLOGICAL AND SURGICAL CONSIDERATIONS.

## HISTOPATHOLOGY OF CEREBRAL TISSUE.

Histologically, cerebral tissue is composed of neurons—the functioning cells of the brain, the most highly specialized tissue in the body. Between the neurons is the supporting tissue of the brain, the neuroglia—the glial cells and fibres. The neuroglia plays only an inactive part in the nervous mechanism; with the small amount of connective tissue from the pia and alongside the blood channels it forms a framework, as it were, in which the nerve cells functionate.

Under stress, such as infection, certain cells of the neuroglia take on amœboid activity and are instrumental in the removal of waste products from the brain.[1]

Following the law that the more highly specialized the cell the more it depends upon nutrition for its life, and the less able it is to regenerate itself, nerve cells die easily and do not regenerate; glial tissue, on the other hand, while more resistant, regenerates. The regeneration of glial tissue, however, plays no active part in the restoration of nerve function. Thus, in traumatic epilepsy, if unrelieved at an early date, a general gliosis, or proliferation of the glial tissue, occurs, in the presence of which any operative procedure for the cure of the epilepsy is useless.

## FACTORS PECULIAR TO CEREBRAL TISSUE WHICH DEMAND SURGICAL RECOGNITION.

Upon opening the dura the following factors peculiar to cerebral tissue are encountered:

(1) In the normal state the dural envelope is completely filled by its contents. During pathological processes, inflammatory or traumatic, the bulk of the intradural content is greater than can be contained within the dural envelope without alteration and compression. Normally, the intracranial tension, being of circulatory origin and varying with the tension of the cerebral veins and capil-

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[1] Alzheimer, Alois: Beiträge zur Kenntniss der Pathologischen Neuroglia und ihrer Beziehungen zu den Abbauvorgängen im Nervengewebe; *Histologische und Histopathologische Arbeit über Grosshirnrinde;* Jena, March, 1910.

laries, is variable in degree; in the dog the brain functions within a wide range of intracranial pressure [2] from zero to 50 mm. Hg. In the presence of a localized intracranial suppuration (abscess) additional space within the dural envelope is obtained by (a) obliteration of the venous sinuses; (b) displacement of the cerebral tissue, not only of the affected but also of the unaffected hemisphere; and (c) obliteration of the cerebro-spinal fluid circulatory system spaces. These factors, by interfering with the delicate circulatory systems—hemic, cerebro-spinal fluid, and intracellular—increase the liability to extension of infection even before the appearance of actual compression of the cerebral tissue.

(2) The over-filling of the dural envelope is partially due to the cerebro-spinal fluid system. In abscess, not only is the amount of the cerebro-spinal fluid increased from the irritation, but there is an associated internal hydrocephalus due to diminished exit of cerebro-spinal fluid by the cortex, and definite obstruction in the main cerebro-spinal fluid circulatory system by displacement of the hemispheres.

(3) The blood vessels of the brain are endothelial tubes, easily ruptured. Every drop of blood effused into the cerebral tissue causes shock by over-stimulation of the cerebral cells. The writer has seen paralysis lasting for days and convulsions continuing for hours as the result of small hemorrhages and pressure on the cortex caused by subdural injections of an autogenous serum.

(4) Injury to cerebral tissue, no matter how slight, has a deeply depressing effect upon the organism as a whole.

(5) Death of ganglionic cells and neurons (the functioning cells of the brain) is not followed by regeneration.

(6) Cerebral tissue, the seat of infection, does not tolerate manipulation and injury as well as normal cerebral tissue; the suppurative process with the associated compression of cerebral tissue so lowers its resistance that slight trauma may occasion extensive œdema or encephalitis. This is of the greatest surgical importance.

(7) The micro-organisms of an encapsulated brain abscess generally are of low virulence while within the abscess cavity; but placed upon the normal piarachnoid they may cause a rapidly fatal suppurative meningitis.

(8) After the evacuation of the brain it must be realized that the piarachnoid and cerebral tissue must be entirely and firmly covered by dura, bone, fascia or skin, as failure to provide a tight

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[2] Hill, Leonard: "Physiology and Pathology of the Cerebral Circulation"; London, 1896.

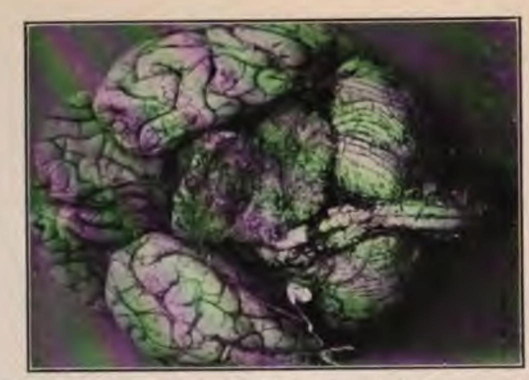

FIG. 2.—Brain, with large tumor, which presented few symptoms for over a year after decompression. Death 2½ years after operation. (This case at no time developed a papillœdema.)

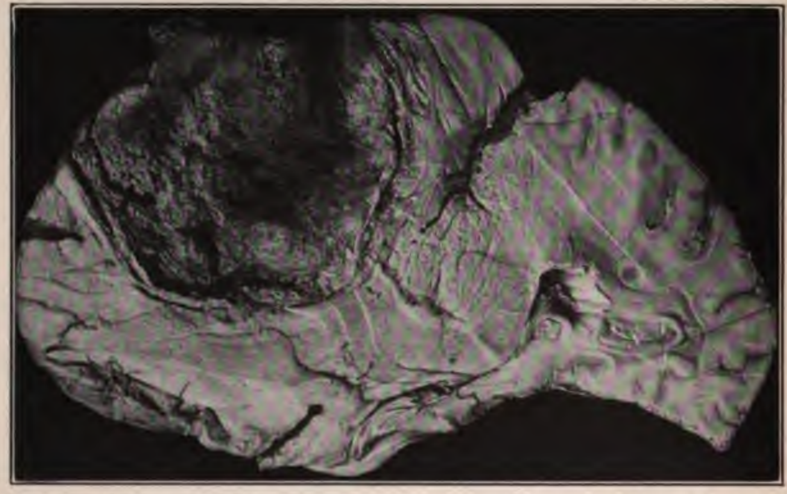

FIG. 3.—Brain, with large tumor. Spontaneous decompression, followed by marked improvement in symptoms.

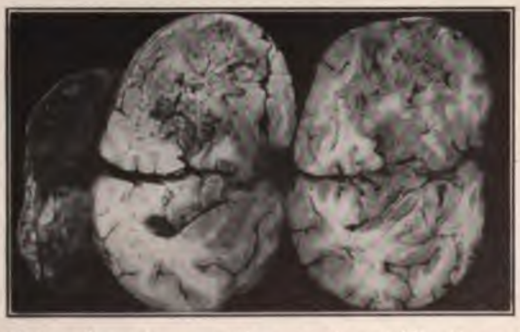

FIG. 4.—Sections of cerebrum with extensive infiltrating glioma. Decompression followed by such relief of symptoms that one year after operation patient, through deception, successfully passed an "inspection" for industrial life insurance.

covering in the presence even of mild suppuration, with associated increased intracranial pressure from œdema, frequently is followed by fungating hernia, which almost invariably terminates in a fatal meningitis. (Witness the fungating hernia following a small "leak" from improper closure or stitch abscess, in a subtemporal decompression.)

### ACUTE ŒDEMA FROM TRAUMA OF OPERATION: SUDDEN INCREASE WITHIN CONTENTS OF DURAL ENVELOPE.

Sudden death may result from acute œdema of the brain, without marked gross changes, as is frequently shown at post mortem.

CASE III, P.

Labyrinthitis. Male, 58 years of age. Undoubtedly serous meningitis, as shown by papillœdema, and excessive pressure of fluid from lumbar puncture, but no micro-organisms in cerebro-spinal fluid. *Operation:* long chiseling process; repeated shocks. Death in coma eighteen hours after operation. *Autopsy:* cortical contents flattened from pressure against skull; excessive amount of cerebro-spinal fluid; no micro-organisms.

This case illustrates a condition which is but an exaggeration of the acute œdema of the brain which occurs with acute abscess, or as the result of trauma.

Cases are not uncommon in which death rapidly follows a cerebral trauma or hemorrhage into a slowly growing glioma, in which at post-mortem, the amount of the hemorrhagic infiltration into the brain is not sufficient, of itself, to cause death, were it not for the rapidity of its effusion.

CASE IV, L.

Glioma. Adult male. Always well. Several months previously headache, followed by convulsion and irregular attacks of "dreamy state." In intervals apparently perfectly well but listless; later irregular headache for two weeks, followed by convulsions, slight aphasia, loss of memory, slight papillœdema; six weeks later suddenly became unconscious. Death within eighteen hours. *Autopsy:* Two gliomatous masses in left temporo-sphenoidal lobe. One, old undergoing cystic degeneration with evidences of former blood extravasation. Second, filled with recent blood extravasation.

In contrast with these cases consider the enormous brain tumors of slow growth, which, when given a vent by decompression, cause few symptoms because the intracranial contents have time to readjust themselves. (See Figs. 2, 3, and 4.)

**Summary.**—The increase in the intracranial contents from suppuration, and the associated protective meningitis, with the possibility of the blocking of the fluid within the ventricles in cerebellar abscess, are all to be considered in the surgical treatment of brain abscess.

In brain abscess the cerebral tissues, as a result of the toxemia from the suppuration and of the increased intracranial pressure, perform their proper functions with difficulty. If, in addition to these, trauma, œdema, or hemorrhage into the cerebral tissue from manipulation should occur, a fatality is apt to result,—a fatality for which the surgeon is largely responsible. How frequently have patients who, prior to operation, were in excellent mental condition, immediately following operation passed into deep coma or active delirium!

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(2) Tylor: In "Pathology of Encephalic Infection in Otitis"; *Brain*, 1912, Vol. XXXV, p. 109.

(3) Homen, E. A.: Experimentelle und Pathologische Beiträge zur Kenntniss der Hirnabszesse; *Arbeiten aus dem Patholog. Institut d. Universität Helsingfors*, 1913, B. I, H. 1-2, S. 1.

(4) Friedmann, M.: Der Hirnabszess (Encephalitis purulenta); *Handbuch der Pathol. Anat. des Nervensystems*, 1904, B. 1, S. 504.

(5) Kölpin: Zur Symptomatologie und pathologischen Anatomie des Hirnabszesses; *Deutsche Zeitschrift für Nervenheilkunde*, 1903-04, B. XXV, S. 365.

(6) Topley, W. C.: A Case of Generalized Streptotrychosis with Extensive Lesions in the Central Nervous System; *Brain*, 1912, Vol. XXXV, p. 26.

(7) Körner, Otto: Die Klinischen Erkrankungen des Hirns, der Hirnhaute und der Blutleiter; 1902.

(8) MacEwen, Wm.: Pyogenic Infective Diseases of the Brain and Spinal Cord; 1893, pp. 972-991.

(9) Politzer, Adam: Diseases of the Ear; Fifth Edition, p. 636.

(10) Wittmaack: Ueber die Beziehungen der pneumatisation Störungen zur Entwickelung endokranieller Komplikationen; *Archiv für Ohrenheilkunde*, B. 97, No. 1.

(11) Hegener, J.: Labyrinthitis und Hirnabszess; *Passow und Schäfer's Beiträge zur Anat., Phys., Pathol., und Therap. des Ohres, der Nase und des Halses*, 1909, B. II, S. 359.

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(13) Westphal, A.: Ueber Gehirnabszesse; *Archiv für Psychiatrie*, 1900, B. XXXIII, S. 206. (Gives detailed pathological and microscopical report with microscopical illustrations.)

(14) Beck, K.: Zur Entstehung und Ausheilung von otitischen Kleinhirnabszessen; *Zeitschrift für Ohrenheilkunde*, B. 64, 1911-12, S. 262. (Gives note on discovering route of infection.)

(15) Essick, C. R.: Pathology of Experimental Traumatic Abscess of the Brain; *Archives Neurology and Psychology*, 1919, Vol. I, p. 673.

(16) Macklin and Macklin: A Study of Brain Repair in the Rat by the Use of Trypan Blue; *Archives of Neurology and Psychiatry*, April, 1920, Vol. 3, p. 353.

(17) Mayman, L.: Zur Pathologie und Klinik der otogenen Grosshirnabszesse; *Münchener med. Wochenschrift*, 1913, No. 2-3, S. 65.

(18) **v. Hibler, E.: Zur Kenntniss der pathogenen Anaeroben. Ein Kleinhirnabszess bei chronischer eiterig-emsiger Otitis, Sinus Thrombose und Carcinom-Entwickelung im rechten Felsenbein; *Centralblatt für Bakteriologie*, 1913, B. 68, S. 257.**

## GENERAL SURGICAL CLASSIFICATION.

Brain abscess, from the standpoint of surgical pathology, constitutes a *group* of surgical problems, separate and distinct, yet so intertwined that they pass imperceptibly from one to another, as nature (or the surgeon) undertakes to limit or eradicate the pathological process. It is by an appreciation of the fundamental pathological differences, by availing himself of favorable conditions, and by the creation of favorable and the avoidance of the creation of unfavorable conditions, that the surgeon may guide a localized intradural suppuration to a successful termination.

From a surgical standpoint, brain abscess may be divided into four general classes, each based on pathological and etiological factors, which are utilizable by the surgeon during the operative treatment.

I—*Chronic Adjacent* (*Secondary*); (temporo-sphenoidal, frontal, or cerebellar) *with a capsule.* The abscess is secondary and adjacent to a known focus of extradural suppuration, such as the mastoid or nasal sinuses, while its slow growth has permitted its limitation by a capsule and has facilitated a readjustment of the disturbed intracranial contents.

II—*Acute Adjacent* (*Secondary*); (temporo-sphenoidal, frontal, or cerebellar) *without limiting membrane.* The rapid formation of the abscess, and its accompanying œdema, have caused a sudden increase in the intracranial contents before the protective mechanism of the brain could have time to erect a macroscopically demonstrable limiting membrane.

III—*Intercurrent* (*Tertiary*) to a gross lesion, namely, sinus thrombosis or progressive osteomyelitis.

IV—*Metastatic,* which includes: (a) Chronic, without a capsule, (b) Chronic, with a capsule, and (c) Acute.

V—*Traumatic.*

## SURGICAL PATHOLOGY.

A large proportion of brain abscesses, (with the exception of the truly traumatic), belong to types I and II, being secondary and adjacent to suppuration within the ear or nose, the infection extending within the dura either by direct continuity of tissue, or what is much more frequent, in my experience, by retrograde thrombophlebitis, with or without an intercurrent sinus thrombosis.

In my series, excluding all truly traumatic cases, out of the first forty-four brain abscesses, forty, or over ninety per cent., belonged to the *secondary adjacent* types, chronic or acute.

Metastatic abscesses originate from embolic septic occlusion of a cerebral vessel, the primary foci being generally far distant from the brain, the most frequent site being the lungs, from a bronchiectasis. Occasionally, a true embolic septic occlusion originates from an infected mastoid or petrous bone, in which case as the septic embolus has passed freely through the circulation prior to its lodgment in the brain, the cerebral invasion is distinctly apoplectiform in onset, while the brain abscess is not adjacent to the ear, but is lodged either in the frontal or occipital lobes, deep within the cerebral tissue, or in the opposite hemisphere.[3] An examination of the clinical and post-mortem histories of the recorded cases of brain abscess, not traumatic, whose situation is not adjacent to the site of infection, demonstrates their embolic origin.[4]

Of the author's forty-four brain abscess cases three were metastatic, one from bronchiectasis, one from suppurative endocarditis, and one, while originating from a sinus thrombosis and mastoiditis, was undoubtedly embolic in origin.[5]

There still remains a small number of so-called "idiopathic" brain abscesses,[6, 7] in which the site of the original suppuration is unknown. The demonstration of the fusiform bacillus as the causative micro-organism in certain of these abscesses and in the tonsils has greatly limited the number. (See Metastatic Abscess, p. 68.)

In types I and II of intracerebral abscess secondary to adjacent suppuration the pathological process is a nutritional death of cerebral tissue following a retrograde thrombophlebitis. While direct extension of the infection through the dura, piarachnoid and brain in the writer's opinion but rarely occasions intracerebral abscess, it is undoubtedly the causative factor in localizing intrapiarachnoid suppuration.

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[3] Lombard, Bloch, A., and Moulouguet, A.: Un cas d'abscès du lobe frontal du côté opposé à une otite suppurée chronique; *Annal des Maladies de l'Oreille*, etc., 1914, Tome 90, p. 749.

[4] Berens, T. P.: Abscess of the Frontal Lobe of the Brain of Otitic Origin; *Trans. Amer. Otol. Soc.*, 1913, Vol. 13, p. 66.

[5] Quimby, Wm. O. G.: *Personal Communication.* (Observed at a post-mortem, a temporo-sphenoidal lobe abscess in the opposite hemisphere to the originating aural suppuration.)

[6] Casamajor, L.: Brain with Double Frontal Abscess; *Medical Record*, 1915, Vol. 87, p. 412. (Report of case and exhibition of specimen autopsy findings, New York Neurological Society meeting December 1, 1914.)

[7] Climenko, H.: A Case of Brain Abscess; *Journal Nervous and Mental Diseases*, 1918, Vol. XLVII, p. 444.

In either case long before the micro-organisms have entered the cerebral tissue,—in one case being confined within the walls of the vessels and in the other because of the continuity of structure—the protective forces of the brain have become active in the affected area. Chief among these, as we have seen, are the leucocytes and endothelial cells from the blood vessels, the cells lining the arachnoid spaces, and the macrophage from the neuroglia, each contributing towards combatting the invading micro-organisms, and the limitation and subsequent repair of the inflammatory septic process by the formation of new connective and the removal of dead tissue. The same applies, to a limited extent, in metastatic abscess because of the temporary limitation of the invading organisms within the lumen of the occluded cell.[8]

In traumatic abscess, on the contrary, the immediate death of cerebral tissue from trauma, associated with the transplantation of micro-organisms in unprepared cerebral tissue, furnishes favorable conditions for the rapid extension of the suppurative process before the limiting protective reactions of the brain can be marshalled; while the associated œdema and hemorrhage of extensive trauma so increases the intracranial pressure that the protective reactions of the brain are actually prevented from playing an active part in the limiting process.

### SURGICAL CLASSIFICATION ACCORDING TO POSITION AND RELATIONSHIP TO THE BRAIN AND ENCIRCLING MEMBRANE.

Intradural abscess may be divided, surgically, according to its position within the brain or piarachnoid, and its relationship with the encasing dura and piarachnoid, *through which its evacuation must* be conducted, into: (*a*) *Intracerebral, without* macroscopical evidence of *cortical involvement;* (*b*) *Intracerebral, with a cortical "stalk"; (c) Meningeal,* either intrapiarachnoid or subdural.

(*a*) *Intracerebral abscess without macroscopical evidence of cortical involvement* originates from retrograde thrombophlebitis, or from perivasculitis from an adjacent area of suppuration—lateral sinus thrombosis, mastoiditis, or sinusitis,—or from the deposit of a septic embolus in metastatic abscess.

(*b*) *Intracerebral abscess with a cortical "stalk,"* probably originates from a combination of direct extension and associated retrograde thrombophlebitis or perivasculitis from an adjacent suppuration. In my opinion the direct extension advances more frequently outward from the abscess cavity itself toward the dura than inward from the dura to the abscess, the "stalk" being always a late manifestation. The "stalk" is nature's effort to evacuate spontaneously an encapsulated intracerebral abscess through the original site of infection. The literature contains numerous instances of the spontaneous evacuation and cure of intracerebral abscess by the development of a stalk followed by a secondary necrosis of the dura.

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[8] See Chapter 9, Protective Mechanism.

As the perivascular spaces of the brain contain cerebro-spinal fluid, not lymph, and histologically and embryologically are not lymph channels, the term "perivascular" should be substituted for the frequently employed and incorrect "lymphatic" path of infection in the brain substance.[9]

(*c*) *Meningeal Abscess,* (either intrapiarachnoid—limited to the meshes of the piarachnoid—or subdural—limited to the subdural space—results from a direct extension from an adjacent infective process—osteitis, phlebitis, extradural abscess or trauma.[10]

As far as the author is aware, subdural and intrapiarachnoid abscess have not previously been differentiated clinically. In a case in which death resulted from leptomeningitis, following the evacuation of a temporo-sphenoidal intracerebral abscess, the post-mortem disclosed a large flat collection of pus and organized fibrin, entirely limited to the subdural space; it was very yellow and resembled the exudate frequently seen covering the appendix, the exudate being about half an inch in thickness and apparently undergoing resolution. It was not entirely a reparative process, however, as it extended underneath the whole frontal lobe of the affected side. It was possibly of traumatic origin from infection of the subdural space during evacuation of the intracerebral abscess, although its appearance and the clinical history would point to its having existed prior to the operation.

The exact site of an abscess in or on the different lobes of the brain depends largely upon its origin and mode of invasion. If the abscess originates from an adjacent suppuration in the ear or nose, it is situated in or on the temporo-sphenoidal lobe, the cerebellum, or the frontal lobe.

An adjacent abscess is almost invariably single, the multiple abscesses reported being generally of traumatic or embolic origin.

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[9] Weed, L. H.: An Anatomical Consideration of the Cerebrospinal Fluid; *Anatomical Record*, May, 1917, Vol. 12, p. 492.

[10] In offering the above classification the author is fully aware of its pathological imperfections. Exact pathological knowledge of the relative frequency of brain abscess from direct extension, from improperly called "lymphatic" infection, from thrombophlebitis, and from septic thrombi by occlusion, is not available since but few microscopical examinations to ascertain the exact route of infection into the brain are recorded in the literature.

An adjacent abscess may be chronic or acute, with or without a distinct limiting membrane, depending upon its age.

If the abscess is of metastatic origin, following a vascular occlusion by a septic embolus or thrombus freed from a distant focus, it may be situated in any part of the cerebrum or cerebellum. It is apt to be multiple, but not so frequently as is generally believed; it is apt to be subcortical, but may be cortical, never, however, possessed of a true stalk; and it may be meningeal. (See Report of Case XVI, T. V., Chapter V, Metastatic Abscess, p. 75.) A metastatic abscess is rarely encapsulated, the limiting membrane being thrombotic, not the formation of new connective tissue.

If the abscess is traumatic, originating from compound fractures of the skull, or from traumatized and infected cerebral tissue during the exploration and evacuation of an adjacent or metastatic abscess, it may be situated in any part of the brain traumatized, a subcortical abscess frequently being associated with a meningeal abscess.

## CLINICAL CLASSIFICATION OF BRAIN ABSCESS.

The importance of a thorough understanding of the origin, mode of infection, relationship with the dura and piarachnoid, and the probable exact site and duration of the intradural suppuration, must be fully appreciated by the surgeon before entering the dura, as each may occasion such variations in the mechanical, pathological and physiological factors as to call for specific surgical recognition.

Exploration into cerebral substance when the abscess is situated entirely within the meshes of the piarachnoid, may result fatally; while the attempted evacuation of a meningeal abscess through a small dural opening—sufficient in an acute intracerebral abscess—is bound to be followed by failure.

If now the surgical types, *Chronic* (*Secondary*) *Adjacent* Abscess *with a capsule; Acute Adjacent* Abscess without limiting membrane; *Metastatic,* and *Traumatic* Abscess; be combined with the specific surgical types:—*Intracerebral* Abscess *without* macroscopical *evidence of cortical involvement; Intracerebral with a cortical "stalk;"* and *Meningeal* Abscess; from the clinical point of view the following varieties in intradural abscess may be recognized surgically, each presenting individual technical problems:—

CHRONIC BRAIN ABSCESS:

1 (*a*), **Intracerebral with Capsule,** without Macroscopical Evidence of Cortical Involvement; Origin; Adjacent, Metastatic, or Traumatic.

This is the most frequent type of adjacent (secondary) abscess, exceptionally of traumatic or metastatic origin.

1 (*b*), **Intracerebral without Limiting Membrane; Origin**; **Metastatic, or** Adjacent.

To this type belong most of the metastatic abscesses, but a certain proportion of otitic and rhinologic abscesses, although of long duration, have no capsule.

2, **Adjacent Intracerebral with Capsule and "Stalk"**; Origin; Adjacent (Secondary), or Traumatic.

The stalk extending through the cortex and attached to the inner surface of the dura over the site of original infection. In exceptional cases the stalk may extend through the dura.

3, **Meningeal**; Origin; Adjacent, Traumatic, or Metastatic.

ACUTE BRAIN ABSCESS:

1. **Intracerebral without Limiting Membrane,** but whose fluid contents are distinctly circumscribed: Origin; Adjacent, Metastatic.
2. **Meningo-Cerebral,** associated with an active surrounding encephalitis: Origin; Traumatic.
3. **Meningeal**; Origin; Adjacent, Traumatic.

Metastatic abscess and abscess originating from sudden vascular occlusion are classified together, and "acute abscess with a stalk" is omitted, the stalk not being macroscopically recognizable and consequently cannot be utilized surgically.

## INTRACEREBRAL ABSCESS WITH A CAPSULE AND "STALK."

In the author's experience abscess with a stalk is not so frequent as intracerebral abscess without macroscopical cortical involvement, although Körner states that forty-two per cent. of all brain abscesses are from direct extension. However, in the reports of 131 cases of brain abscess analyzed by the author, a sinus leading to the abscess was found in but four. The abscess situated in both the cortex and the subcortical tissue is attached by its stalk to an adjacent area of suppuration in either the temporal bone or the accessory sinuses of the nose. It is a chronic process, the entire abscess and stalk being distinctly encapsulated.

There is reason to believe that intracerebral abscess with a stalk is a late stage of intracerebral abscess without macroscopical evidence of cortical involvement. The stalk develops after the formation of the abscess cavity by a continuation of tissue necrosis from the abscess cavity *toward the cortex,* following in reverse order the path of infection originally taken by the causative thrombophlebitis, the nutritional disturbances of the cerebral tissue, plus the pressure of the abscess from above, causing the necrosis.

Those who have viewed through a large dural opening thrombosis of a cortical cerebral artery; who have seen how distinctly the area of improper blood supply is demarked, and yet how normal it appears until compared with the surrounding cortex, can understand how the area surrounding a retrograde thrombophlebitis is the seat of an extension of the suppurative process from the abscess.

The writer's reasons for believing that the stalk is frequently a secondary process after the development of the abscess itself are: that abscesses with stalk are much less frequent than intracerebral abscesses without macroscopical evidence of cortical involvement; that the stalk is found only in old abscesses; that post-mortem examinations show a large number of extensive abscesses with a perfectly normal cortex; and that there is sometimes seen at post-mortem an area of necrosis of the surface of the cerebral tissue, not extending from the dura.

In the abscess with a stalk from direct extension of tissue necrosis, the suppuration having advanced from the adjacent site of infection, firmly uniting the piarachnoid with the inner surface of the dura and extending through the cortex, furnishes a direct route into its interior without injury to the subarachnoid or subdural spaces.

During operation, if the dural opening is large the stalk is easily located by the area of adhesions of the piarachnoid to the dura. This type of abscess, if evacuated early, should give a high percentage of recovery. Many cases of brain abscess have recovered spontaneously by evacuation through the stalk and the formation of a fistula in the dura. This—Nature's method—the establishment of an encapsulated tract through the dura, piarachnoid and cerebral tissue into the abscess cavity—should be one of the first principles to be followed in surgical treatment.

In like manner in adjacent abscess the surgeon should attempt to convert an intracerebral abscess with a limiting membrane but without macroscopical evidence of cortical involvement into a *capsulated* abscess with a stalk; *i.e.,* to convert an acute abscess without limiting membrane into an encapsulated intracerebral abscess.

## INTRACEREBRAL ABSCESS WITHOUT CORTICAL INVOLVEMENT DISCOVERABLE MACROSCOPICALLY.

This type is generally caused by a retrograde thrombophlebitis of a cerebral vessel.[11] Upon opening the dura no evidence of disease is discoverable in the piarachnoid or the cortex, the abscess being situated entirely within the cerebral substance at a depth of from one to four centimeters beyond the cortex, but adjacent to the primary infection.

Although at operation the cortex usually appears normal, in one instance in a metastatic abscess secondary to bronchiectasis the writer found a small thrombosed vessel running from the dura through the arachnoid space, the abscess being located directly under the affected vessel. The author is not familiar with a similar observation during life,[12] but surgically it suggests a careful inspection of all vessels running from the dura into the brain for surrounding infiltration or thrombosis.[13]

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[11] Preysing, H.: Neun Gehirnabszesse im Gefolge von Ohren-und Nasenerkrankungen; *Archiv für Ohrenheilkunde,* 1900-01, B. 51, S. 262.

The peculiar blood supply of the brain accounts for the formation of the abscess entirely beyond the cortex.[14] The blood vessels of the pia run horizontally upon the surface of the cortex and anastomose freely; consequently, thrombosis of a pial vessel but infrequently interferes with the nutrition of the cortex sufficiently to produce extensive local necrosis. On entering the cerebral tissue, however, the blood vessels run vertically and become terminal; infective thrombosis of a cerebral vessel, therefore, is rapidly followed by local nutritional death of cerebral tissue with abscess formation.

The cerebral necrosis from lack of proper nutrition, following infective vascular occlusion of a terminal and intracerebral vessel, explains the frequency with which intracerebral abscesses grow inward toward the ventricle—rupture into a ventricle being a frequent termination—and the futility of leaving the dura open "so that the pus may follow the line of least resistance," in case of failure to locate an abscess.

"The line of least resistance" in nutritional death is really inward. If a capsule is not formed the brain offers but little resistance until the abscess approaches the ventricle, when, according to Miodowski,[15] the choroid plexus may take on a definite protective action, becoming thickened by several layers of exudate. (This observation has not been confirmed in the writer's experience, although he has repeatedly examined the choroid plexus at postmortems.)

## SUBDURAL ABSCESS.[16]

Subdural abscess rarely exists except as a traumatic complication of intracerebral abscess. The literature contains but a few references to collections of pus confined to the subdural space.[17] The author's own experience is limited to the case referred to on page 73, which, it is believed, originated from the exploration and evacuation of an intracerebral abscess through a small dural opening.

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[12] MacEwen, W.: "Pyogenic Infective Disease of the Brain and Spinal Cord"; 1893, p. 90, Fig. 39. (Depicts a thrombophlebitis over the area of a brain abscess at postmortem.)

[13] The vessels that normally enter the dura directly from the cerebral cortex are comparatively few and consequently may be rapidly investigated.

[14] Piersol: Textbook of Human Anatomy, III Edition, 1911, p. 1206.

[15] Miodowski, F.: Beiträge zur Pathogenese und pathologischen Histologie des Hirnabszesses; *Archiv für Ohrenheilkunde,* 1908, B. 77, S. 239.

[16] There exists confusion in the anatomical nomenclature. Some authors regard the smooth, glistening lining of the dura as the parietal layer of the arachnoid, and consequently would call it the arachnoid instead of the subdural space. (Santée, H. E.: "Anatomy of the Brain and Spinal Cord," 1915, p. 9.)

The frequency of subdural abscess as a complication of experimentally produced traumatic intracerebral abscess would make it appear that trauma is the usual cause.[18, 19, 20]

## METASTATIC ABSCESS.

This is usually situated within the white substance of the brain, secondary to septic vascular occlusion, the infection having been brought from a distant focus by the circulation. All possess the two characteristics of metastatic abscess—the situation away from the site of original infection, and an apoplectic onset.

Metastatic brain abscesses are frequently secondary to bronchiecstasis, pyemia, and endocarditis, but are rarely associated with aural suppuration, although a personal experience has convinced the writer of their occurrence.[21, 22] From an operative standpoint metastatic abscess belongs to the group having no macroscopical cortical involvement; the frequency of multiple metastatic brain abscesses, however, should be recognized. (See Metastatic Abscess, p. 68.)

## TRAUMATIC BRAIN ABSCESS.

Prior to the development of otology and rhinology, and before the recognition of aural and nasal suppuration as the cause of adjacent brain abscess, trauma was regarded as the most frequent cause of intradural suppuration. Although its importance has been much neglected in recent years, an examination of the recorded cases demonstrates that, in the presence of a chronic or acute adjacent suppuration, trauma still may be regarded as playing an active part. Many

[17] Streit, Herman: Weitere Beiträge zur Histologie und Pathologie der Meningitis und Sinusthrombosis; *Archiv für Ohrenheilkunde,* 1912, Bd. 89, S. 177. (Gives analyses of reported cases.)

[18] Essick, C. R.: Pathology of Experimental Traumatic Abscess of the Brain; *Arch. Neurol. and Psychol.,* 1919, I, p. 673. (States that during the experimental production of brain abscess, "in one-third of the animals the infection entered the subdural space, forming a subdural abscess.")

[19] Heine, B.: Zur Kenntniss der subduralen Eiterungen. *Beiträge zur Ohrenheilkunde; Festschrift Lucae,* Julius Springer, Berlin, 1905. (Contains an excellent colored illustration of a subdural abscess.)

[20] Hinsberg, v.: Ueber dem Infections Mechanismus bei Meningitis und Stirnhöhleneiterung; *Verhandlungen der Deutsche Otol. Gesellschaft,* 1901, S. 191. (Gives microscopical findings of path of invasion by blood vessels and lodgment in subdural space, with microscopical illustrations.)

[21] Jobson, G. B.: Contralateral Otitic Brain Abscess; *Laryngoscope,* Vol. 25, 1915, p. 7.

[22] Eagleton, W. P.: Report of a Case of Brain Abscess Resulting from an Infective Vascular Occlusion of Otitic Origin, with Involvement of the Internal Capsule, clinically Simulating Cerebral Apoplexy, and Presenting Unusual Psychic (toxic delirium) Phenomena. Operation—Recovery—Subsequent History. (Reported at the annual meeting of the Medical Society of New Jersey, held at Spring Lake, N. J., June, 1915.)

cases of chronic otitis first present cerebral symptoms following a blow or fall on the head. (See Case XII, W. K., Chapter IV, page 60.) Likewise, in many recorded cases the first symptoms of brain abscess followed a surgical manipulation, such as a radical operation, on a neighboring bony structure. Such cases are so numerous that the inference is forced that the trauma of the operation was the direct cause of the brain abscess, or lighted into activity a long dormant intracerebral suppuration. (See Analysis of post mortem examination of cerebellar abscess, p. 105.)

Traumatic brain abscess may be defined as a collection of pus, the result of death of cerebral tissue in the presence of infection, the tissue death resulting not from the infection, as in secondary adjacent brain abscess, but from trauma coincident with the infection.

Traumatic infective encephalitis passes imperceptibly into traumatic brain abscess. Infective encephalitis is a diffuse septic process, whereas traumatic brain abscess is a partially—but only partially—limited one. The difficulty of limiting the process is the distinguishing feature between traumatic and adjacent abscess. In the latter the protective mechanism of the brain has been marshalled, the bulwarks of resistance have been prepared before the actual entrance of the invading micro-organisms. In the former the trauma, by killing cerebral tissue, has furnished a most favorable medium for the growth of micro-organisms before provision for combating them has been made; and the secondary œdema following the trauma,—by blocking the nutrient channels of the brain, by the induction of thrombosis and hemorrhage into its substance, and by increasing the brain's bulk and thus causing compression—has established a vicious circle which not only favors extension of the infection, but actually paralyzes all attempts at limitation.

These specific factors of traumatic abscess (death of cerebral tissue prior to preparation; secondary œdema with associated hemorrhage and thrombosis; increase in the brain's bulk, all causing compression with further death of tissue, and paralysis of the brain's protective mechanism) should forewarn the surgeon because an encapsulated adjacent abscess—which has been fully walled off—may be converted during operation into a traumatic abscess with its associated encephalitis.

## MENINGEAL ABSCESS.

**Intrapiarachnoid.**—This type, the result of direct infection through the dura, is at first a protective process of the piarachnoid in

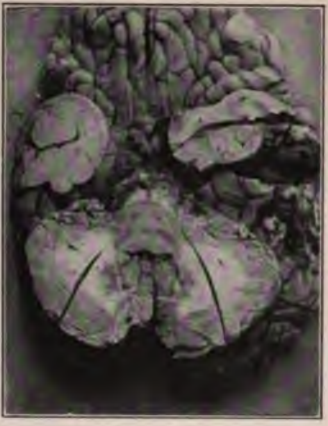

FIG. 5 (a)

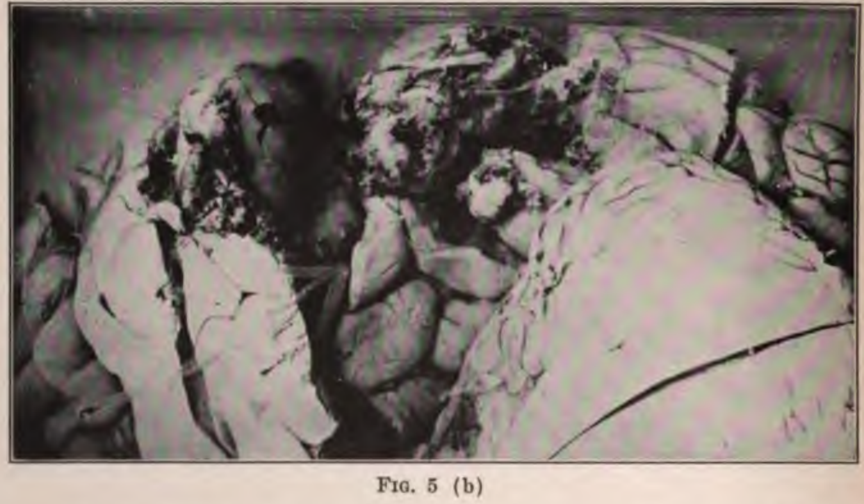

FIG. 5 (b)

FIG. 5 (a) and (b).—Brain abscess situated in meshes of piarachnoid; superficial necrosis from pressure. Exploration and evacuation through small opening. Death from meningitis. Autopsy disclosed a further brain necrosis and abscess contiguous to dural opening. Osteoplastic flap and dural incision would have exposed the entire intrapiarachnoid abscess and prevented the cutting off by brain pressure of the undiscovered portion.

its attempt to prevent general infection of the meninges.[23] The delicate cells of the piarachnoid take on great activity when approached by infection, and offer a very efficient barrier to the invasion of micro-organisms from either the dural or the cerebral surface. While our knowledge of the function of the cells lining the subarachnoid spaces is still very meagre, they play an active part in the brain's protective mechanism. In the presence of an adjacent suppuration, a large collection of cerebro-spinal fluid is frequently localized around the infected area, which later may become a localized collection of pus.

As our present knowledge but rarely admits of a positive differential diagnosis between cortical and subcortical lesions, the surgeon, before exploring the brain substance, should examine the brain surface in all directions accessible to the dural opening. If the abscess is situated within the meshes of the piarachnoid, exploration into the brain tissue is not only unnecessary, but may seriously militate against the patient's recovery. In three cases after an unsuccessful exploration of the cerebral substance, the post-mortem disclosed an abscess localized entirely within the meshes of the piarachnoid. In each instance I failed to locate the intrapial abscess because I was working through an insufficient opening. All of these cases, though ending fatally, were favorable cases for recovery, had they been properly treated. (See Fig. 5 a and b.) These experiences convince me that exploration through an intact dura, as so frequently practised, is fundamentally wrong.

## GENERAL SURGICAL PRINCIPLES.

Clinical experience and experimental observation abundantly teach that one of the prime objects in the surgical treatment of intracerebral abscess, secondary to adjacent suppuration, is the utilization of the already organized protective reactions of the brain. Surgical manipulations should be so conducted as not to paralyze this protective mechanism. If the operative trauma to the brain is not the minimum necessary to meet the individual requirement—the eradication and obliteration of the abscess in an encapsulated abscess with a stalk—the complete or partial evacuation in an encapsulated intracerebral abscess, or simply the partial evacuation of the abscess for the relief of increased intracranial pressure in an acute abscess, (a localized collection of pus—localized either actually if a capsule has been formed, or potentially if the abscess is acute) will convert such abscess into a traumatic abscess, with almost inevitable rapid extension of the infection, cerebral necrosis, and death.

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[23] Klippel, D.: Méningites Chroniques: *Nouveau Traité de Médecine de Therapeutique,* 1912. Tome 35, p. 274.

**Surgical Factors.**—The factors to be considered in surgically approaching a brain abscess are: (1) the duration of the cerebral suppuration, (2) the presence or absence of a limiting membrane, and (3) the extent of the disturbances in the brain substance, outside the abscess. The different pathological processes not only call for variations in the operative technic to be adopted, but markedly influence the possibility of successful surgical intervention.

Irrespective of the pathological condition and because of the factors peculiar to cerebral tissue, the surgeon on entering the dura must endeavor to prevent all injury to the brain from its own pressure; to lessen, as far as possible, injury to the brain tissue by manipulation thereby converting the abscess into an unlocalized traumatic process; to prevent the spread of the infection to tissues as yet not infected, especially the meshes of the piarachnoid; to control compression, which of itself favors extension of the suppurative process, and to convert an unfavorable into a favorable type of abscess whenever this is possible.

**General Technic.**—Slow, painstaking work, from the first nick in the dura to its complete closure, is of primary importance. The intradural work should be performed with as much delicacy and attention to detail as are required during the extraction of a cataract. Each drop of blood effused into the piarachnoid or the cerebral tissues, each separate trauma, hemorrhagic or manipulative, lessens the probability of the patient's recovery.

Essick[24] believes that there is experimental evidence that whole blood limits the spread of infection to the meninges. If this is substantiated, it will revolutionize our ideas of the treatment of the subarachnoid spaces during the evacuation of brain abscess.

The experiments of Flexner[25] and Weed,[26] however, demonstrate that any disturbance of the protective mechanism of the brain—the withdrawal of cerebrospinal fluid, the production of an aseptic meningitis by the injection of sera, or the infusion of blood within the subarachnoid spaces—greatly predispose to a general infection of the meninges in the presence of a blood stream infected by certain micro-organisms. Of course Essick's experiments approach much nearer to the conditions present during the evacuation of a brain abscess than the induced blood stream infections of Weed and Flexner. The latter, however, demonstrate how delicate the protective mechanism of the nervous system is and how easily it is disturbed, while clinical experience teaches that hemorrhage into the subdural spaces in the presence of infection actually facilitated its spread. Until Essick's observation has been amplified, the surgeon must be careful to avoid all effusion of blood within the meninges or brain.

[24] Essick, C. R.: Pathology of Experimental Traumatic Abscess of the Brain; *Arch. Neurol. and Psychol.*, 1919, I., p. 673.

[25] Flexner, S., and Amoss, H. L.: The Relation of the Meninges and Choroid Plexus to Poliomyelitic Infection; *Jour. Exper. Med.*, 1917, Vol. XXV, p. 525.

[26] Weed, L. H., Wegeforth, P., Ayer, J. B., and Felton, L. D.: Meningitis Produced by Intravenous Inoculation; *Monographs of Rockefeller Institute for Medical Research*, No. 12, March 25, 1920, pp. 57-112.

### INJURY TO THE BRAIN FROM ITS OWN PRESSURE.

Upon opening the dura excessive internal pressure may cause cerebral herniation, with injury to the arachnoid, associated with slight hemorrhage into its substance. In acute abscess, the tension within the dura is always excessive; consequently, if the dura appears very tense, before opening it a lumbar or ventricular puncture, depending upon whether the suppuration is in the cerebrum or in the cerebellum, may be performed, although the early withdrawal of cerebro-spinal fluid is to be avoided if possible.

#### LUMBAR PUNCTURE; ITS DANGERS:

Lumbar puncture, with the withdrawal of a small amount of fluid prior to the incision of the dura in abscess of the cerebrum, will allow the brain to fall away from the dura so that it can be incised without injury to its contents. The performance of lumbar puncture is not without danger, especially if there be an abscess below the tentorium, as, by the removal of the cushion of the cerebro-spinal fluid from below, the increased pressure above may drive the medulla through the foramen magnum, herniating it, with resulting immediate paralysis of the respiratory centre. I have seen death on the table following a lumbar puncture; while the fluid was still flowing, the patient stopped breathing, and although the heart continued to beat for some time, during which artificial respiration was continuously performed, the patient from the time of the accident gave no evidence of voluntary respiration.

#### VENTRICULAR PUNCTURE:

In cerebellar abscess the intracranial pressure is not only increased by the abscess, but also by an internal hydrocephalus,—a usual complication of cerebellar abscess. (See Appendix, Part IV.)

In abscess of the brain, with its complicating internal hydrocephalus, the increased intracranial pressure may obliterate the lumen of the large venous sinuses, the blood, ordinarily carried by the sinuses, leaving the skull by the normally small diploic vessels. Extreme venous obstruction, so frequent in tumors of the posterior fossa, is not encountered, however, during operations for cerebellar abscess, as death will have supervened long before an advanced stage is reached.

Sufficient venous obstruction to obliterate the lateral sinus of the affected side, giving rise to a mistaken diagnosis of a sinus thrombosis, has been observed by me during operation. When venous obstruction is present the removal of bone over the cerebellum may be accompanied by considerable hemorrhage, and the opening of the dura without immediate prolapse of the brain and rupture of the piarachnoid will be impossible, especially if only one hemisphere is exposed. If, however, a ventricular puncture is performed before the dura is opened, the sinuses again become patent, the bleeding stops and the cerebellum drops back, permitting dural incision without prolapse of the brain.

#### Technic of Incision of the Dura:

To avoid possible injury to the piarachnoid, Cushing originally caught and elevated the dura with two fine silk ligatures, cutting between them. Later he employed a small, sharp, half-curved membranous cataract hook for the primary opening. By either method the dura can be incised without injury to the piarachnoid. When the dura is not excessively tense, it can generally be safely incised by cutting superficially with a knife, scraping a little from side to side, and then cutting a little deeper and scraping again. In several instances, however, I have injured the piarachnoid during this procedure. The small dural opening may be enlarged safely by cutting on a flat brain spatula, the dural vessels being "clipped" by Cushing's silver wire[27] or ligated before incision.

## CLASSIFICATION OF BRAIN ABSCESS ACCORDING TO LOCATION.

The anatomical and physiological relationships of suppuration in the frontal, temporo-sphenoidal, occipital lobes and cerebellum occasion great differences, not only in symptomatology, but in surgical considerations. A useful clinical classification, however, is into: Abscess of the Middle Fossa (Temporo-sphenoidal), Frontal lobe abscess and Cerebellar abscess. The occipital lobe is in such intimate relationship with the temporo-sphenoidal lobe that surgically they are considered together, suppuration confined to the occipital lobe alone occurring only in abscess of metastatic or traumatic origin.

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[27] Cushing, Harvey: The Control of Bleeding in Operation for Brain Tumor, with the Description of Silver "Clips" for the Occlusion of Vessels Inaccessible to the Ligature; *Trans. Amer. Surg. Assoc.*, 1911, XXIX, p. 389; *Annals of Surgery*, 1911, Vol. LIV, pp. 1-19.

# CHAPTER IV.

# ABSCESS OF MIDDLE FOSSA.

## ADJACENT (SECONDARY) ABSCESS OF THE MIDDLE FOSSA—TEMPORO-SPHENOIDAL.

### LOCATION; PATHOLOGICAL AND ANATOMICAL CONSIDERATIONS:

During the early stages of brain abscess, the symptoms as a rule are so vague that they do not warrant a positive diagnosis of intracranial suppuration, much less its definite localization. Consequently any operative procedure which may be undertaken at an early stage must be largely exploratory and must be so conducted that in case of failure to locate the suppuration as little damage as possible, immediate or remote, will be caused by the exploration.

The usual sites of intradural suppuration of the middle fossa secondary to aural disease, are,

(1) in the meshes of the piarachnoid, directly above the tegmen of either the antrum or the middle ear; such an abscess is probably preceded by, and results from, an extensive but still localized meningitis, a pathological condition which lumbar puncture and post-mortems lead the author to believe is of greater frequency than is generally recognized:[1]

(2) in the substance of the temporo-sphenoidal lobe itself, in the second temporo-sphenoidal convolution, or

(3) an extension from one or other of the above.

## INTERCURRENT (TERTIARY) ABSCESS OF THE MIDDLE FOSSA, SECONDARY TO THROMBOSIS OF THE SUPERIOR PETROSAL SINUS OR A DURAL VEIN:[2]

As thrombosis of the lateral sinus is often the immediate mode of transmission to distant portions of the cerebellum, away from the petrous, so thrombosis of the superior petrosal is in rare cases the transmitting agent of the temporo-sphenoidal lobe; for it is to be remembered that one of the main venous efferents of the basal portion of the brain—one of the medio-cerebral veins, often called the temporal sinus—empties into the superior petrosal sinus.[3, 4]

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[1] Brieger: In Blau's *Encyclopedie der Ohrenheilkunde*, 1900, S. 245.

[2] See Case XXVIII; Chapter XI, page 182.

### ADJACENT MENINGEAL ABSCESS (INTRAPIARACHNOID OR SUBDURAL) OF THE MIDDLE FOSSA.[5]

Post-mortem examinations lead the writer to believe that an abscess primarily situated in the meshes of the piarachnoid, the result of direct extension through the tegmen, is apt to remain confined to the meshes of the piarachnoid. As it increases in size, the intrapiarachnoid abscess frequently involves the cerebral cortex, by direct pressure causing "superficial brain necrosis," but never resulting in an intracerebral abscess.

### Localized Serous (Protective) Meningitis.

To this group belong the cases of so-called "serous meningitis," with localizing symptoms, aphasia, paralysis, etc., which recover after the removal of the focus of infection within the temporal bone without opening the dura. Serous meningitis is probably primarily a sterile, localized collection of cerebro-spinal fluid of toxic origin, which later may become easily infected—the primary stage of an intra-piarachnoid or subdural abscess.[6, 7, 8, 9]

The experimental work of Essick, in which the presence of bacteria in the adjacent tissues without invasion of the subarachnoid space itself causes a large increase in the amount of cerebro-spinal fluid and leucocytes, explains the frequency of serous meningitis and its operative treatment, *viz.*—

(a) The removal of the adjacent infected area of the mastoid cells or the petrous pyramid, and (b), the prevention of compression by decompression through a clean area, lumbar puncture or the evacuation of the fluid by incision of the dura through the infected area. The latter method is frequently successful, but it allows the evacuation of only a small quantity of fluid, with the liability of infection of an uninfected area.

[3] Browning, W.: The Veins of the Brain and its Envelopes: Their Anatomy and Bearing on the Intracranial Circulation; Monograph, Brooklyn, 1884.

[4] Whitehead, A. L.: Some Points in the Diagnosis of the Complications of Temporal Bone Disease, Based upon a Study of 135 Fatal Cases; *Journal of Laryngology, Otology and Rhinology*, 1906, Vol. XXI, p. 269.

[5] See p. 17, Surgical Classification of Brain Abscess.

[6] Boenninghaus, Georg: "Die Meningitis Serosa Acuta"; Wiesbaden, Verlag von J. F. Bergman, 1897. (Reviews cases reported prior to 1897.)

[7] Warrington, W. B.: Intra-cranial Serous Effusions of Inflammatory Origin, Meningitis, or Ependymitis Serosa; *Quarterly Journal of Medicine*, January, 1914, Vol. 7, No. 26, p. 93. (Reviews the literature.)

[8] Knapp, A.: Serous Meningitis; *Archives of Otology*, 1906, No. 1, p. 10.

[9] Emerson, Linn: A Case of Serious Meningitis Mistaken for Brain Abscess; Operation; Recovery; Reprint from *Laryngoscope*, July, 1906.

CASE V, R. B.

**Sinus Thrombosis.** Male. Chronic otitis media, left ear. Frequent attacks of pain and headache; chills. Admitted to hospital suffering from mastoiditis, left side. Mastoidectomy done. Sinus thrombosis discovered; jugular not ligated because of condition of patient; thought to be dying. Recurrence of symptoms.

**Operation:** Ten days after mastoid operation, ligation of jugular. On opening vein it was found to contain free pus for a distance of about three inches, running down below the clavicle. The pus was not in the fascia around the vein, but was in the vein itself.

Immediately after the second operation the man complained of dizziness. Ten days later chill; violent headache, repeated at regular intervals during the day. Patient gradually became violent from pain, which passed as suddenly as it came. Had two chills. Marked papillœdema of both eyes. Labyrinth dead. Spontaneous deviation of both hands to right. Fell to right. No spontaneous nystagmus. Slight area of anesthesia along third branch of trifacial. One attack of vomiting. Lumbar puncture revealed cloudy fluid under pressure; cell count 160; increased globulin.

Spontaneous deviation to right and falling to right, both away from lesion, pointed to involvement of cerebellum over posterior surface of petrous portion of temporal bone. This was also confirmed by cerebro-spinal fluid, which gave symptoms of protective meningitis.

Cerebellum exposed in front of sinus. Incision of dura through sinus region; in front of this evacuated large amount of cerebro-spinal fluid.

**Diagnosis:** Serous meningitis. Uneventful recovery.

CASE VI, J. G.

**Aseptic Meningitis.** Male, 26 years of age. Right ear had been discharging for past three years, with attacks of pain, which, however, were not severe. Never any dizziness except when he had grippe about one year before coming to hospital; but did not think it was associated with any exacerbation of his ear trouble. About three weeks before admission to hospital had an ulcerated tooth; after extraction the pain continued for a time and then subsided. At the same time he began having pain in his ear, and dizziness; pain in ear was not steady—it was sometimes severe and sometimes not. It was associated with pain in the head in the right temporal region, running forward toward the frontal region. During these three weeks he had dizziness—ran into things but did not fall. Swayed at times, but could not tell to which side he was inclined to fall. Had two attacks of vomiting, not influenced by food.

When first seen had no facial paralysis, but three or four days later was found to have a right-sided facial paralysis, and the discharge in the ear was very much increased. Examination of his ear showed a large mass of polyp coming from roof. Had a slight convergent strabismus; was totally deaf in right ear; complained of chilly sensations; no patella reflexes; no sense of taste.

**Operation:** A radical operation was done, following which the cochlea was opened and the vestibule entered. The external semicircular canal had necrosed through. The dura over the internal auditory meatus was opened, evacuating cerebro-spinal fluid; no increased tension. For several weeks patient had considerable temperature, pain in his head, stiff neck, and high cell count. Some weeks later patient discharged sequestra of cochlea. It was then seen that protective meningitis had followed operation. Uneventful recovery. (The case is recorded in *The Laryngoscope*, for January, 1922.)

**Clinical Diagnosis.**—With a high temperature without chills but with symptoms of compression, such as papillœdema, if repeated blood cultures have been negative or the sinus has been explored and found to be uninfected, or if the infection has been thoroughly eliminated, and especially if cytologically an aseptic meningitis is present—see Case V, R. B.—we are warranted in suspecting an aseptic meningitis of the irritation type. In which case, before exploration within the dura it is justifiable—(1) to thoroughly evacuate all infected areas, mastoid, labyrinth or sinus, and (2) to control the increased intracranial pressure by decompression through a clean area, ventricular puncture or lumbar puncture. The procedure to be adopted for the control of compression depends somewhat upon the site of the suppuration, for it must always be doubtful if an intracerebral abscess is not also present. If the labyrinth and sinus are both intact the decompression should be a subtemporal one, while if either the labyrinth or sinus is known to be involved, and consequently a cerebellar abscess is possibly present, a ventricular puncture or a cerebellar decompression should be performed. The danger of a lumbar puncture causing sudden death from respiratory paralysis in cerebellar abscess is so great that it should only be resorted to in case a ventricular puncture for some reason is not advisable.

### EXTENSION OF INTRAPIARACHNOID ABSCESS.

In its growth a localized meningeal collection of pus, starting from just above the tegmen, usually extends externally along the under surface of the temporo-sphenoidal lobe; then upward and forward, along the external surface of the temporo-sphenoidal lobe in which direction it may travel a considerable distance. A meningeal abscess so situated is always very shallow, due to the firmness with which the temporo-sphenoidal lobe is held to the floor and sides of he middle fossa by the weight of the brain, the exit of the nerves and vessels toward the median line, and the veins entering the lateral sinus. An abscess so situated is frequently divided into several partments communicating with each other—the so-called "oyster-ed abscess."

Rarely the localized meningeal collection of pus extends directly ward, inward and upward, along the upper surface of the ten-ium cerebelli, below the occipital lobe. When the abscess is situ-d above the tentorium, the purulent collection is apt to be very in amount but of very thin consistency, being in reality simply localized collection of purulent cerebro-spinal fluid, not

In evacuating a meningeal abscess on the external surface of the temporo-sphenoidal lobe, a part of the abscess may be easily overlooked because the internal pressure of the brain forces it firmly against the edge of the dural flap, thus cutting the abscess in two. In meningeal abscess over the tegmen, a similar accident is not infrequent, the brain descending before the abscess is completely evacuated. This is illustrated by the following case:

CASE VII. J. J. C.

**Brain Abscess.** Steam fitter, well developed and nourished, admitted to hospital November 18, 1912. Diagnosis: Brain Abscess.

Two months previously, patient "got cold in ears" while swimming, and two days later became almost totally deaf first in right, then in left ear. Abscess developed in both ears and broke in right. No discharge from ears. The right ear gradually got better, but pain continued in both ears. About two weeks before admission to hospital pain increased in left ear, and patient became very deaf in that ear. There was never any discharge from either ear. Eight days before the patient was seen, intense pain developed in head and in left ear, with dizziness, and the man was obliged to stop work. Tympanic membrane lanced. For three days previous to admission had been almost totally deaf in left ear, with intense pain; also had peculiar frontal headache and pain in eyes. He suddenly felt something break in head which sounded in left ear like cannon shot, and since explosion had word deafness; vomiting three days before and during explosion. There was a previous history of pneumonia in childhood, and typhoid 11 or 12 years ago. Used neither tobacco nor alcohol.

Examination revealed right sided hemianopsia, paresis of right arm, and Wernicke's aphasia. Could recognize family and his own doctor, but could not understand their names. There was word deafness, slight dizziness, absence of right-sided abdominal reflexes, exaggerated knee-jerks, especially the right, exaggerated plantar reflexes, and tendency to ankle clonus on right side; no Oppenheim, no Babinski, no nystagmus, but increased P. R. on right side. Tongue very coated; no tremors. Left ear: could hear watch on contact; deaf to aerial conduction. Right ear: could hear watch at 2 inches.

**Operation.**—A large flap was made above the left ear and the skull exposed and trephined; mastoid not opened. Some pus was found in the external auditory canal. Dura found to be adherent and covered with granulations; no pus in dural cavity. Upon exposing the temporo-sphenoidal area of the brain an oyster-shaped abscess was discovered in posterior portion of lobe, with distinct limiting membrane. Brain surface was irrigated with warm saline, the dura closed and rubber drain inserted. The scalp was closed with interrupted sutures of plain catgut.

The following day patient appeared confused, but was able to recognize names of objects shown to him. Temperature 102°. A lumbar puncture was done and about 300 c.c. of fluid withdrawn under tension. Paresis of right arm still present, and probably hemianopsia of right side. Patient unable to talk, and upon attempting to read printed numbers tried to make them; usually made them inverted.

Nine days later patient had two severe convulsions, lasting about five minutes each. Temporo-sphenoidal region again explored, but no abscess found. Rubber tissue placed over brain and a drain passed through scalp. Patient then turned on abdomen and incision made in median line of neck down to base of skull, bone chiselled away and medulla exposed. Dural and basal cisterns opened and rubber drain inserted into the latter. Gauze drain inserted down to base of wound.

The next day patient had convulsion which lasted one minute, and on following day convulsion and chill. The day after, he had convulsion at 2.30 A.M. which lasted one minute, and another at 4.45 P.M., with incontinence. Death occurred at 1.00 A.M., on the thirteenth day after operation.

Post-mortem examination showed that this man originally had infection of the left middle ear, which was followed by the extradural abscess disclosed at operation. The abscess was walled off from the dura by firm granulations, and was followed by an intradural, extrapial abscess, which at operation appeared to be intracerebral; the covering, however, proved to be simply a thick membrane. Patient subsequently developed suppurative meningitis. The infection was evidently one of low virulence, as no bacteria were found in the cerebro-spinal fluid or in the pus examined. Local collections of pus at post-mortem exhibited a tendency to recede under the occipital lobe in the position of normal drainage from gravity. Nothing abnormal was found in the ethmoidal, sphenoidal, or lateral sinuses, or in the opposite ear.

## ADJACENT INTRACEREBRAL ABSCESS OF THE TEMPORO-SPHENOIDAL LOBE WITHOUT MACROSCOPICAL MANIFESTATIONS OF CORTICAL INVOLVEMENT.

Abscess of the temporo-sphenoidal lobe, without macroscopical evidence of cortical involvement is the most frequent variety of brain abscess. It develops either from a retrograde thrombophlebitis, or from direct extension of tissue suppuration from the beginning of the mastoid antrum or of the middle ear. When it originates from a retrograde thrombophlebitis because of the anatomical vascular condition, it is situated in or near the second temporo-sphenoidal convolution, which, being a silent area, produces no outstanding localizing symptoms on the right side in right-handed patients; but if situated on the left side it frequently produces a "naming" aphasia, which latter may at first be of but transient duration and of imperfect extent.

Adjacent abscess may, however, be situated entirely within the occipital lobe—(H. Knapp)—the author having seen one such case where the abscess was secondary to a lateral sinus thrombosis, or thrombosis of the superior petrosal sinus. Its situation may also be entirely removed from the temporo-sphenoidal lobe.

This type of abscess may attain great size, having a tendency to extend inward from nutritional disturbances. Rupture into the ventricle is a frequent termination.

## TEMPORO-SPHENOIDAL ABSCESS WITH A STALK.

"Abscess with a stalk" is located almost invariably in the second convolution of the temporo-sphenoidal lobe, directly above the tegmen in the cerebral substance, generally from one-half to one and a half inches above the tegmen. (Figs. 6 and 7.) The external sur-

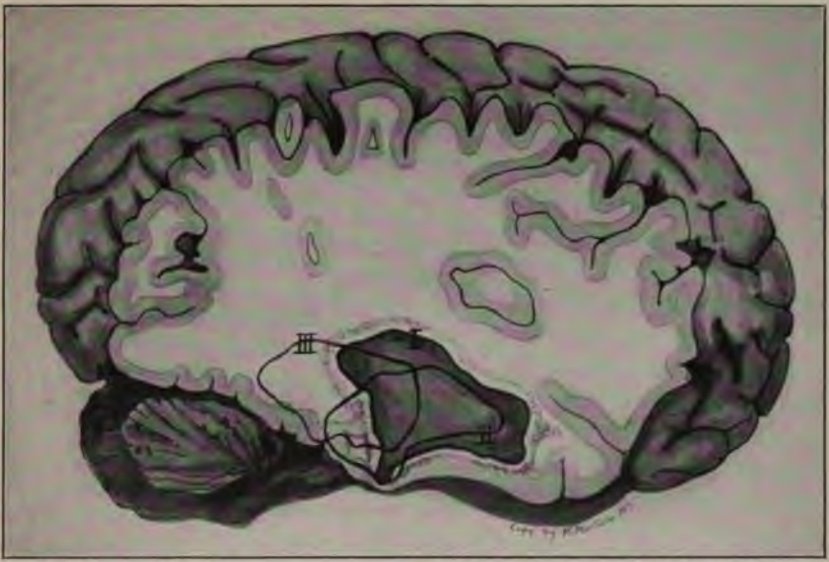


FIG. 6.—Position of three temporo-sphenoidal abscesses with a stalk, above the tegmen tympani and antrum, showing uniform position of adjacent abscess. Illustration copied from Preysing drawing in Körner's "Die otitischen Erkrankungen des Hirns, der Hirnhäute und der Blutleiter," 3d Edition, 1902.

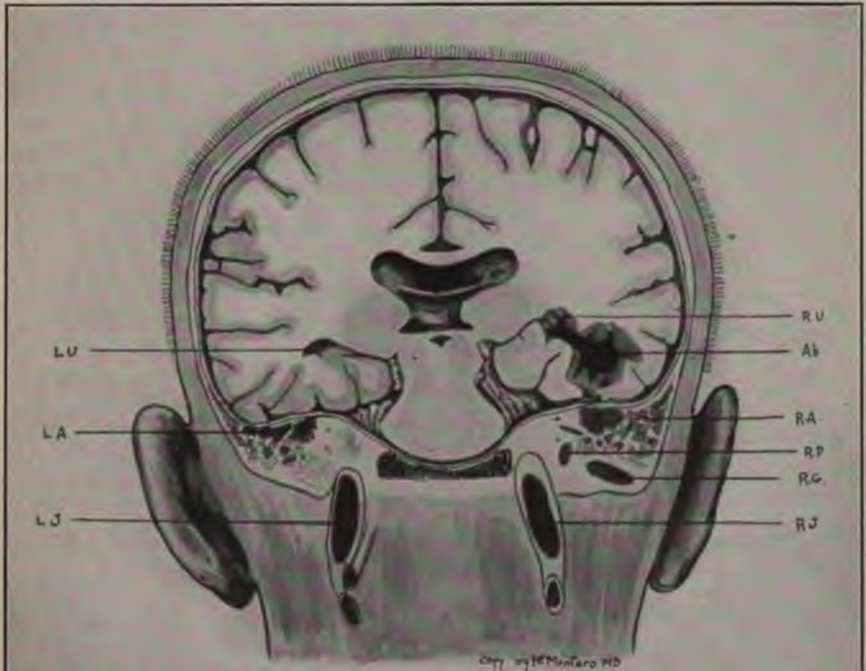


FIG. 7.—Horizontal section of head with temporo-sphenoidal abscess with stalk, showing uniform position of adjacent abscess within cortex, low down in temporo-sphenoidal lobe, above antrum or middle ear. Illustration copied from Preysing drawing in Körner's "Die otitischen Erkrankungen des Hirns, der Hirnhäute und der Blutleiter," 3d Edition, 1902.

face of the dura is frequently adherent to the bone and covered with granulations, and the dural inner surface is always united to the brain.

### CONSIDERATIONS PRIOR TO OPERATION FOR ADJACENT (SECONDARY) ABSCESS OF MIDDLE FOSSA.

A diagnosis of a probable adjacent (secondary) temporo-sphenoidal lobe abscess having been made, (based upon the presence or history of an adjacent suppuration within the mastoid or petrous portion of the temporal bone; the general symptomatology of the intracranial suppuration; localizing symptoms varying between negation and the pathognomic "naming" aphasia; elimination of cerebellar suppuration), a definite opinion must be formed prior to operation, whether the abscess is chronic with a probable capsule, or acute without limiting membrane. This is of prime importance, as the technic to be adopted is fundamentally different, and failure properly to visualize the surroundings of the abscess may lead to an irreparable blunder.

The operation must not be simply an effort to find and evacuate pus, irrespective of its surroundings; it must be a procedure adapted to existing conditions, the most important being the presence or absence of a limiting membrane. This is in opposition to the dictum that "brain abscess, as pus in other parts of the body, is to be treated by free drainage." Reviewing my many failures in the light of post-mortem findings, I am convinced that to avail himself of every advantage, the surgeon must be fairly sure before he operates whether a capsule does or does not limit the abscess.

Histories of two cases of temporo-sphenoidal lobe abscess improperly treated because of incorrect diagnosis as to pressure or absence of capsule.

CASE VIII.

**Temporo-sphenoidal Lobe Abscess**—Chronic. A. M. M. Male. Progressive muscular atrophy of left side, beginning about a year before admission to hospital. Four weeks before admission he began to have neuralgia on left side of face, with pain in teeth, continuing after paracentesis of membrana tympani of left side. Ear continued to discharge. One week before admission became unable to talk. On admission had typical "naming aphasia." Mild papillœdema of left eye. Occlusion of left aural canal. Slight paralysis of lower portion of right side of face, seen chiefly on emotional movement. Right-sided hemianopsia; right hand slightly weak; no Babinski; no clonus; apparently deaf in left ear. Cerebration rather slow. Lumbar puncture gave apparently normal fluid; culture revealed microörganisms.

**Operation.**—Believing it to be an acute abscess, temporo-sphenoidal region exposed over an area of an inch and a half in diameter. A small dural incision made through which the brain protruded. Gangrenous. Free pus evacuated through small needle. No capsule felt. Following operation slow pulse, patient in good condition, apparently disorientated.

**Autopsy Findings.**—On removing the skullcap by the Washburn method the whole anterior lower inner surface of the anterior fossa between dura and brain was found to be covered with a yellowish exudate which looked like chicken-fat, the thickest portion being several millimeters thick; it was smooth, glistening, and extremely yellow. This covered the whole of the frontal lobe of the left side. The abscess itself was in the temporo-sphenoidal lobe, was about one inch in diameter and had a beginning membrane, the margins of which were becoming sharply defined. Its inner surface was red, undoubtedly from hemorrhages which had undergone absorption. There was no free pus in the abscess. There were many areas of slough or moisture in the abscess which, as a whole, was round with very few pockets. The area around the abscess was yellow, taking on the character of the exudate. There was typical lepto-meningitis in several parts of the brain. The lateral ventricle of the left side was much displaced over and beyond the median line and was slightly distended. The opposite lateral ventricle was markedly distended. The left middle ear showed an extensive area of degeneration, bluish-yellow from acute necrosis. The anterior wall of the middle ear was apparently entirely gone, leaving a fistula. The inferior petrosal sinus contained an exudate in the portion before it enters the cavernous sinus; the cavernous sinus was free. The mastoid cells were not markedly involved; the ethmoids and sphenoid not involved. On removing the dura over the tegmen the bone looked bare—no communication was seen between the area of the abscess and the tegmen. No adhesions between the dura and the brain.

**Comment.**—In making a small dural incision and trying to pass a small Whiting's encephaloscope through the small opening nothing was accomplished except to incite a traumatic encephalitis, as was shown afterwards by the gangrenous hernia. The man had an acute abscess which should have been simply evacuated through a large or a small opening in the dura, but nothing else should have been done. On evacuation the pus was first watery and then flocculent. This should have warned us that this was an acute abscess. The opening made was large enough to permit herniation but too small to permit inspection.

Three days after operation the pulse was only 88, although the temperature was as high as 104°. The patient was restless. Systolic blood pressure was 100. Five days after the operation the pulse was 68, temperature 102°, and the patient apparently better. After operation there was marked papillœdema of both eyes—not present before operation—indicating compression.

CASE IX.

**Temporo-sphenoidal Abscess** (Chronic). G. W. Male, seven years of age. Admitted to hospital March 1, 1920. Running ear three months before; mastoid operation February 8, 1920.

**Operation:** Operated on March 3, 1920, for temporo-sphenoidal abscess. Evacuation through a small opening in the dura with a very small needle. No pus was withdrawn, but a large quantity of clear fluid having the appearance of cerebro-spinal fluid evacuated. On introduction of the needle resistance was encountered, but on the assumption that we were dealing with an acute abscess, this was not recognized as due to the capsule of a chronic abscess. Strabismus was noted at this time. The patient did very well for three days following operation. The strabismus almost entirely disappeared, but recurred. Six days after operation paralysis of the rectus of the opposite eye appeared and the patient became sleepy and drowsy, growing gradually worse until the fourteenth day after operation, when death occurred.

**Autopsy Findings.**—Very extensive encephalitis surrounded an encapsulated abscess which was adherent over the tegmen. The encephalitic tissue was soft,

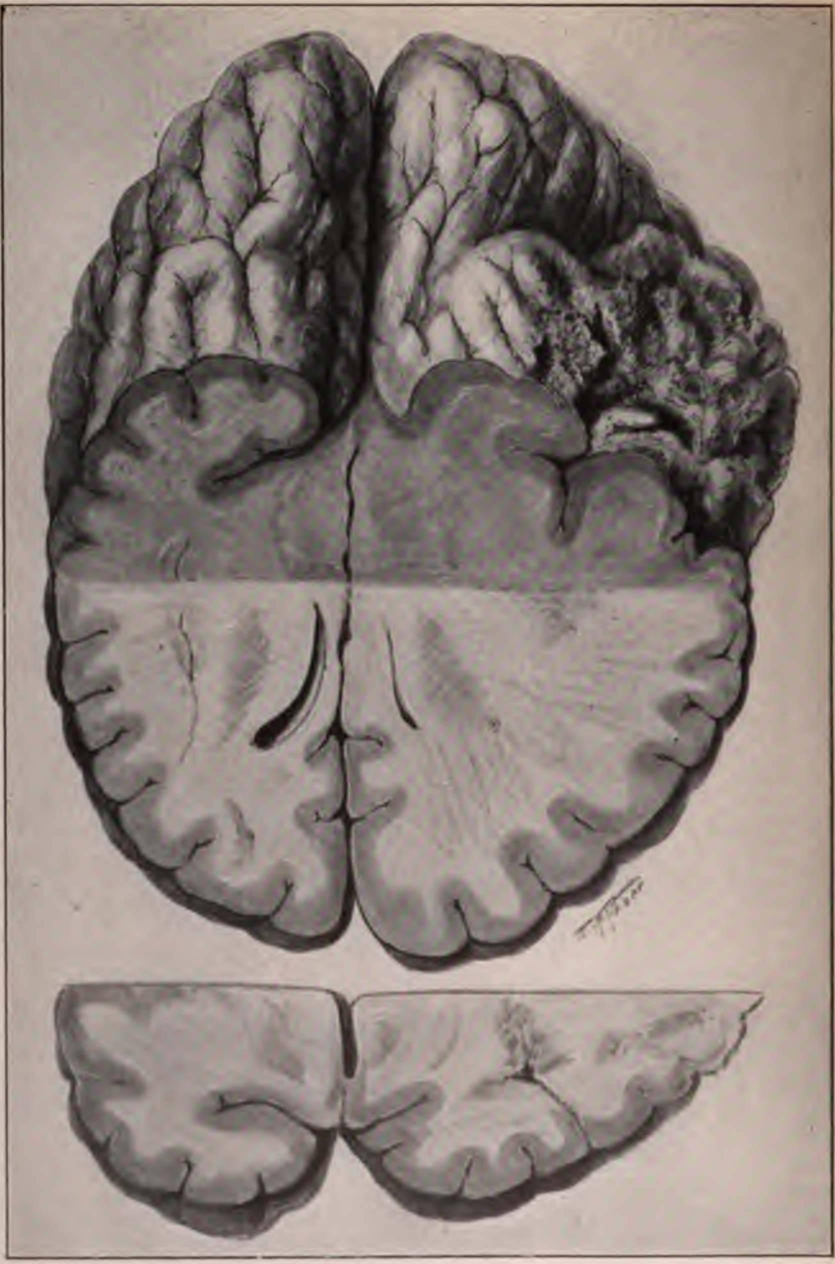


FIG. 8.—Temporo-sphenoidal lobe abscess cavity with displacement of hemisphere beyond median line. Showing increase in size of brain bulk. Death from extension of suppurative process and compression after several weeks of apparent freedom from symptoms. Abscess erroneously treated as an acute abscess by small opening when it was a chronic abscess with capsule.

with numerous hemorrhages. The abscess had approached the cortex in many places, causing gangrenous areas. The capsule was distinct, firm, and in some places several millimeters thick. It was so firm that it was covered with blood vessels and by taking hold of it the whole abscess was lifted out intact. The pus surrounding the capsule was thin, such as is developed from an acute encephalitis, while that inside the capsule was thick, such as is seen in chronic brain abscess. (Fig. 8.)

**Comment.**—From the clinical course after operation and from the autopsy findings this was evidently a case of chronic (encapsulated) brain abscess, which was treated as an acute abscess. The death of the patient was probably the outcome of this failure to differentiate the acute from the chronic condition, and of the traumatizing of the brain tissue in the effort to drain the abscess, the procedure followed being likewise the outcome of this error in diagnosis.

At the time of operation, it will be remembered, a large quantity of perfectly clear fluid was drawn away which looked like cerebro-spinal fluid and was found to contain streptococci. At that time it was thought that this might be a large collection of cerebro-spinal fluid outside the brain, the abscess being intra-arachnoid. The clearness of the fluid also suggested that the abscess was acute. It is unfortunate that it was not recognized that the resistance encountered in the efforts to evacuate the abscess was due to the encapsulation of a chronic abscess, and that this encapsulation accounted for the absence of chill after the patient was admitted to the hospital. The chill probably had occurred at the time of the mastoid operation, seven weeks before, at which time, doubtless, the abscess had begun to form. The autopsy showed that the clear fluid drawn off at the time of the abscess operation was probably from the top of the abscess, the pus having settled below. The exploring needle being so small, only the clear fluid could pass through it. When the encapsulation was encountered during the exploration, a larger needle should have been used, on the theory that the abscess was chronic and the pus too thick to pass through a small needle.

This case may be contrasted in that respect with that of Mrs. H., Case XIII, Chapter Four, p. 63, in which the abscess was easily evacuated through a small needle. However, the question arises whether, at the time of the operation on the boy, it could have been determined that the abscess was chronic and not acute.

In the diagnosis reliance must be placed upon the difference between the symptoms of compression from chronic abscess and those of brain destruction from acute abscess. In an acute abscess there probably is brain destruction going on, with symptoms of sepsis, as shown in Case XIII, Mrs. H., to which reference has just been made. One of the symptoms of compression is slowing of the pulse. The fact that this boy had an irregular pulse should have called attention to the probability of the extension of the process, but not necessarily to compression. The increase in the optic neuritis was also significant. The temperature was of little assistance. A second lumbar puncture might have been of assistance in the diagnosis.

An error in technic involved the improper anchorage of the stylet tube in the capsule. The cotton must be placed in pledgets close up to the tube itself, touching it in all its parts, building a pyramid which comes from the side of the apex where the tube is. Over this must be placed a dressing and the bandage must go around the neck. Failure to observe these two essential proceedings caused the tube to become displaced in this case and also in Case XIII.

Furthermore, failure to place the drainage tube *within* the abscess capsule, which, as stated, was not recognized as such at the time of subsequent dressing and exposure of the wound, was an error in technic, giving rise in manipulation to undue traumatization of the tissues and setting up the encephalitis found at autopsy. In other words, this chronic brain abscess was converted into an acute abscess which caused the patient's death. If the compression caused by the chronic abscess had been relieved by a decompression operation, the encephalitis might not have developed, the chronic process would not have been converted into an acute one, and the final outcome of the case might have been very different.

### PRINCIPLES OF OPERATIVE TREATMENT.

**Exposure.**—Exposure of the under surface of the temporosphenoidal lobe through the roof of the tympanum and antrum, followed by puncture of the dura and brain, and introduction of drainage material, has resulted in a larger proportion of recorded cures than any other method. Were it possible to make a positive diagnosis of an encapsulated intracerebral abscess of the temporo-sphenoidal lobe, with a stalk, this would be the method of election, as the surgeon thus enters the abscess through the walled-off area of primary infection at its nearest approach to the cortex. Such a diagnosis is infrequent except on finding a fistulous tract during or subsequent to mastoid operation. Many such cases are on record.

In the greater proportion of cases, if the surgeon is to operate early enough to offer a fair chance of recovery, the operation must be largely explorative. Exposure and exploration through the tegmen has the following objections: (a) It is through an infected field; if the abscess is not located, death from meningitis is almost inevitable. (b) The exposed area is so limited that an intrapiarachnoid abscess may be entirely overlooked; if exposed, its ramifications are obliterated by the brain pressure. (c) The surgeon, seeing a few drops of pus, cannot know it to be part of an extended, thin layer of extra-cerebral suppuration, but realizing that a larger collection of pus must exist to account for the symptoms, he is tempted to explore the brain substance, creating a traumatic intra-cerebral suppuration. (d) An intra-cerebral abscess without macroscopical evidence of cortical involvement, an abscess with a stalk, and a meningeal abscess cannot be differentiated, and consequently cannot be differently treated. (e) Compression cannot be provided for except by enlargement of the dural opening into a clean area after an operation in a highly infected area—the reverse of a properly planned surgical procedure.

*Exposure for Suspected Localized Intra-dural Suppuration of the Middle Fossa.*—The operative procedure is planned to form an osteoplastic flap sufficiently large to admit of complete inspection and exploration of localized intra-dural suppuration of the cerebrum, without involving the infected middle ear and mastoid, and if an abscess is not located, to allow complete aseptic closure of the dura and replacement of the flap. (See Frontispiece.) If an abscess is

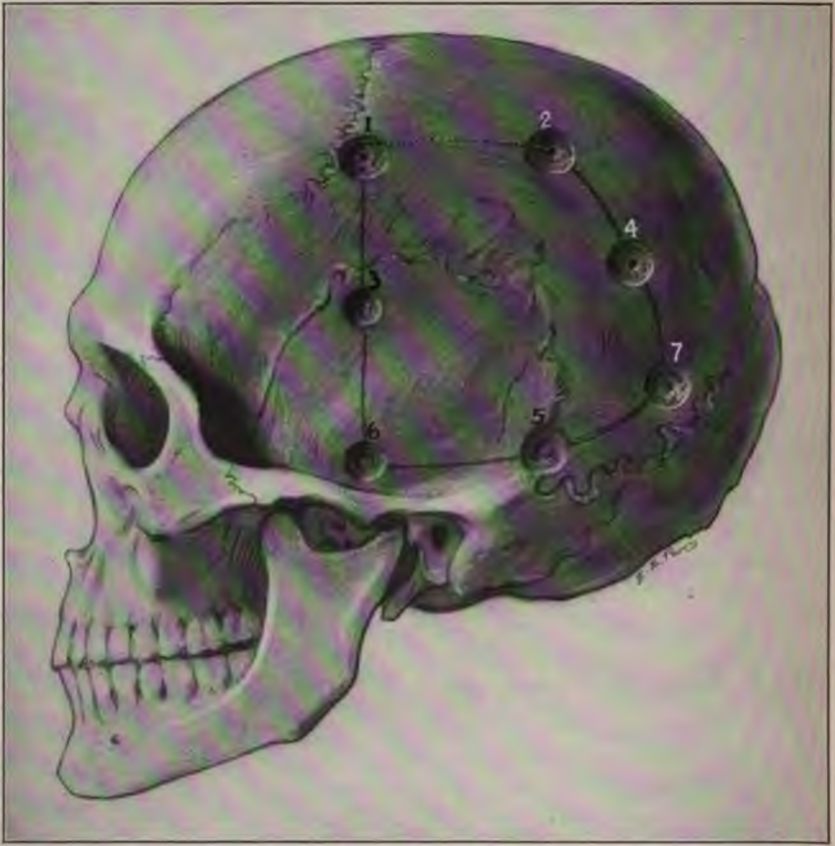


FIG. 9.—Outline for formation of osteoplastic flap for exploration for suspected abscess of the temporo-sphenoidal lobe.

located, especially one contiguous to the middle ear, the flap permits of drainage through the middle ear, the primary route of infection.

### THE OSTEOPLASTIC FLAP FOR EXPLORATION FOR ABSCESS OF MIDDLE FOSSA.

*See Frontispiece.*

**Technic.**—Preparation. The osteoplastic flap, irregularly trapezoidal in shape, has its base upwards. The limits of the proposed flap are first outlined by a very superficial skin incision; the area is covered by moist gauze, and Lake's semi-circular incision is then made. This incision, which surrounds the upper one-half of the ear, begins posterior to the auricle in the auricular fold in front of the mastoid, hugs the attachment of the cartilage to the skin, and ends anteriorly at the lower level of the epitragus in front of the ear. The incision, carried through the fascia and muscle to the bone, allows of a forcible reflection of the external ear, with an evulsion of the membranous canal downward.

A small piece of cotton to which is attached a black string is now placed in the space between the exposed superior auricular bony wall and the reflected canal in order to absorb whatever purulent secretion may be expressed from the middle ear by pressure.

After covering with another layer of moist gauze, the reflected aural flap may be held in position downward by a narrow, flexible tourniquet, so placed that its upper edge exposes the superior wall of the external auditory canal, and consequently is below the level of that portion of the middle fossa formed by the superior surface of the petrous pyramid. Cushing's canopy is then applied, limiting the operative area.

If, as is usual, the mastoid has previously been opened, the whole procedure is considerably complicated, as the walling off of the infected area of the previous operation, Lake's incision, the downward reflection, and the filling of the whole cavity with iodoform gauze, must be treated as an entirely separate procedure preparatory to the aseptic exposure of the brain above.

Technic of Formation of Bone Flap.[10]—The skin incision for the lower border is from three and a half to four inches in length. It begins one inch in front of and three-quarters of an inch above the external auditory meatus, on an imaginary line perpendicular to the center of the eminentia articularis, runs horizontally backward for two inches to an imaginary line perpendicular to the posterior border of the base of the mastoid, and then turns sharply upward at an angle of 30 degrees and runs one and one-half inches to the posterior inferior angle of the parietal bone, just above the parieto-occipital suture. The anterior incision, three and one-half to four inches in length, is at right angles to the anterior section of the inferior border. (Figure 9.) The skull is perforated at both superior angles (Holes 1 and 2); then at the center of both anterior and posterior borders (Holes 3 and 4). The superior halves of the anterior and posterior borders, (Line "a," between holes 1-3, and Line "b," between holes 2-4),

[10] See Chapter Two, p. 10. The formation of the flap is described in detail because, while osteoplastic resections in this region have been frequent (Krause, *Surgery of the Brain*, Vol. 3, p. 900, English edition, 1912), the author is not aware that a replaceable osteoplastic flap has previously been devised which automatically aims at utilizing all available space as near the floor of the middle fossa as possible without injury to the sinus and without entrance into the infected areas of the middle ear and mastoid. During the last few years the author has made many of these bone flaps, and must confess that the majority have become infected when an abscess has been located because of their close proximity to an infected ear. However, in a case in which an abscess is not located the whole flap can generally be saved even if a local area of infection may have occurred.

With this disadvantage the osteoplastic flap has served a most useful purpose and the author believes that with strict observance of technic it will commend itself for all cases of suspected temporo-sphenoidal lobe abscess.

are then formed by the Gigli saw, making the bevel outward, after which, ***under the skin***, the bone of the base of the flap, (Line "c," between holes 1-2), is partially sawed so as to insure the flap breaking properly. *The formation of the lower border of the flap, which is three and one-half inches in length, is the most important, the object being to have it reach as near as possible to the floor of the middle fossa, where the principal investigation will be held.*

The bone of the lower border is perforated, (Hole 5), at the junction of the anterior (horizontal) and posterior (elevated) portions in the squamous portion of the temporal, directly over the root of the mastoid, about one inch behind and three-quarters of an inch above the external auditory meatus, and above and slightly in front of the root of the temporal ridge, which here makes a sharp bend upward. The bone over the anterior inferior angle, (3-5-6), being deep under the temporal muscle, can be sacrificed as well as the bone below it as far as the floor of the middle fossa. The remainder of the anterior border is then cut with a Gigli saw. The posterior half of the inferior border is then elevated to avoid the knee of the lateral sinus, which here rises very high. The posterior inferior angle, (Hole 7), one and one-half inches above Reid's line, is in the inferior angle of the parietal bone, just above the parieto-occipital sutures, two inches behind the end of the horizontal portion of the inferior border of the anterior section. In making the posterior inferior angle, (Angle 4-7-5), hemorrhage is frequently encountered from external diploic vessels running directly into the sinus, which may necessitate the rapid completion and elevation of the flap to control the hemorrhage. Consequently the perforation at the posterior inferior angle, (Hole 7), and its connection with the anterior sector of the inferior border, (Line "d," between holes 5-7), and the posterior border, (Line "e," between holes 7-4), is left until all other parts of the flap have been made. (Fig. 10, Elevation of Flap.)

**Inspection of Brain within the Dura.**—Free incision of the dura exposes the external surface of the temporo-sphenoidal lobe, frequently the site of oyster-shaped localized intra-arachnoid abscess.

### Herniation of Brain upon Incision of Dura in Temporo-sphenoidal Abscess.

While moderate herniation of the brain occurs immediately upon incision of the dura, in my experience the excessive uncontrollable herniation associated with rupture of the piarachnoid and hemorrhage, which frequently accompanies exposure of the cerebellum, especially when but one cerebellar hemisphere is exposed, does not occur. Making a large dural opening greatly lessens the chief danger from the herniation, viz., rupture of the piarachnoid. If threatened, this calls for reduction of the internal cerebral pressure by lumbar puncture. However, if the abscess is large and so situated that it compresses the ventricles with displacement of the hemisphere, lumbar puncture may liberate but a few drops of cerebrospinal fluid apparently not under tension.

**Obliteration of Homolateral Ventricle.**—As the abscess increases in size, the lateral ventricle gradually becomes compressed;

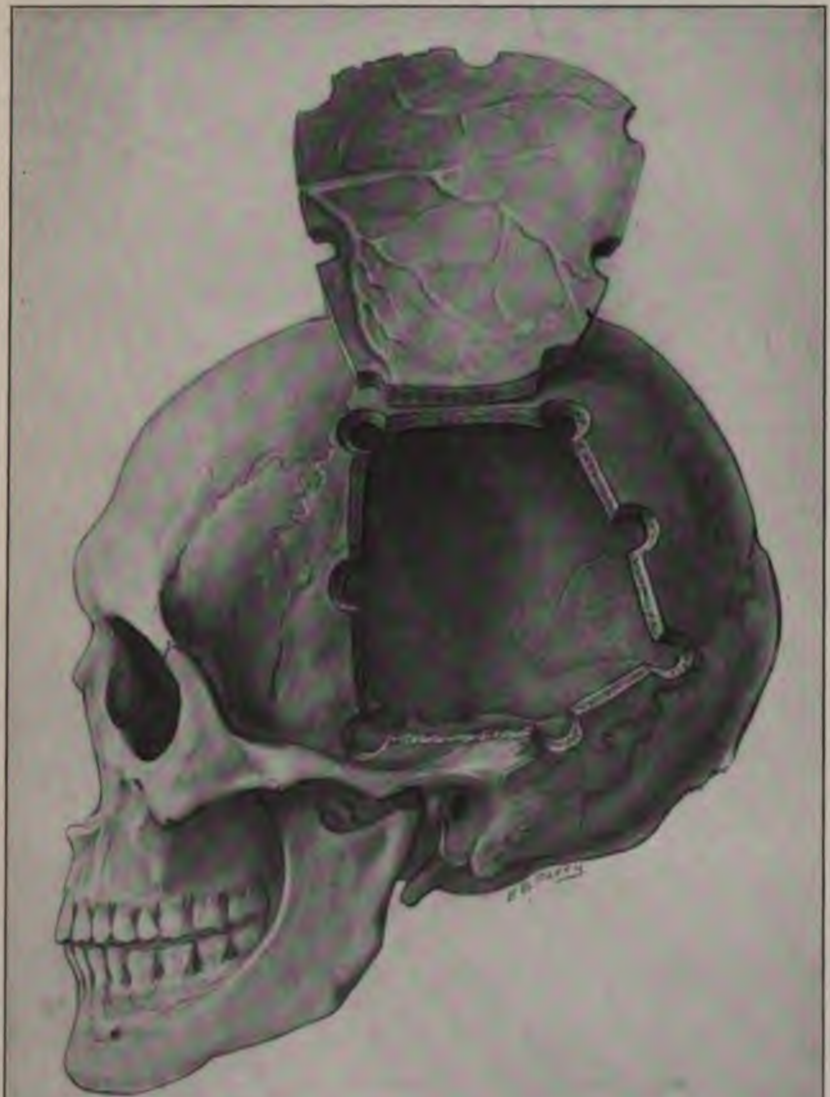


FIG. 10.—Bone flap elevated. (See Frontispiece.)

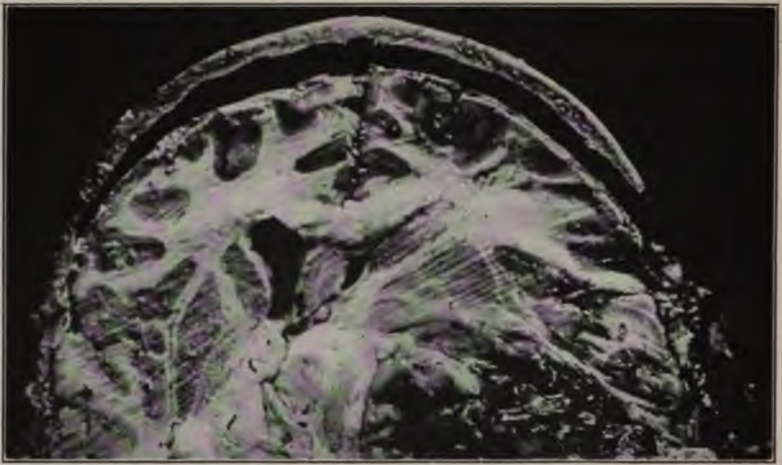
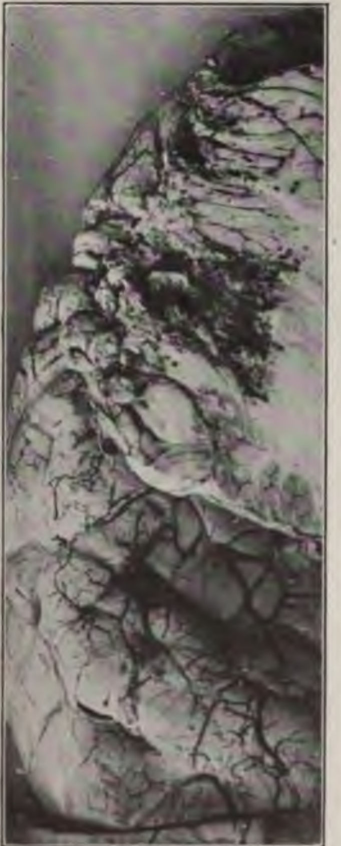


FIG. 11.—Section of brain in skull, showing temporo-sphenoidal hemorrhagic infiltration and herniation, with displacement of homolateral hemisphere beyond the median line and almost complete obliteration of lateral ventricle although a large dural opening had been made. Prior to the dural opening the displacement and the obliteration must have been extreme.



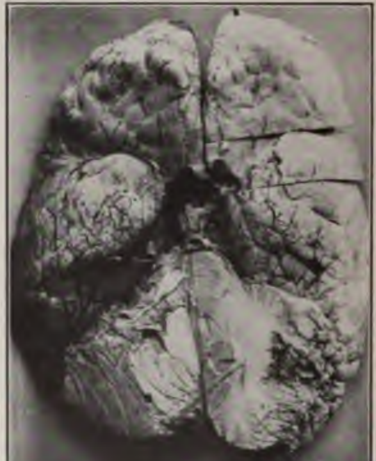


FIG. 13 (a)

FIGS. 13 (a) and (b).—Acute cerebellar abscess in lateral lobe of cerebellum—a frequent position when secondary to sinus thrombosis. During evacuation pus passed over piarachnoid; prior to evacuation a careful examination of cerebrospinal fluid gave no evidence of suppurating meningitis; immediately following operation fulminating meningitis. Death within thirty-six hours.

FIG. 13 (b)

this is followed by a partial displacement of the cerebral hemisphere beyond the median line, probably with actual obliteration of the aqueduct of Sylvius.

Obliteration of the ventricle of the affected side, with displacement of the hemisphere beyond the median line by abscess, is a matter of great surgical importance to which attention has not hitherto been directed. Cushing has long recognized that subcortical tumors frequently obliterate the ventricle of the affected side, preventing its location by ventricular puncture. Of the frequency of obliteration of the lateral ventricle and partial displacement of the hemisphere the writer has post-mortem evidence in one case of cerebral abscess (Fig. 11), in two cases of subcortical tumor of the temporo-sphenoidal lobe, and, clinically, by repeated failures to find the ventricle on puncture.[11]

The following case demonstrates obstruction in the main cerebro-spinal fluid channel by cerebral abscess, the compression and displacement of the hemisphere probably causing a "block" at the aqueduct of Sylvius and the restoration of the channel by the evacuation of the abscess.

CASE X, C. H.

**Temporo-sphenoidal Lobe Abscess.** Girl. Diagnosis: Abscess of temporo-sphenoidal lobe. On incising dura great herniation of brain. Lumbar puncture liberated but a few drops of cerebro-spinal fluid, dropping slowly at intervals of several seconds. Exploration within brain by canula; partial evacuation of abscess through canula. Upon relief of excessive pressure cerebro-spinal fluid in large quantities under pressure began to flow from the lumbar puncture canula, the partial evacuation of the abscess having removed an obstruction which prevented the free flow of the cerebro-spinal fluid. (Cf. Case XXIV, Chapter VIII, p. 135, Hernia Cerebri Following Temporo-sphenoidal Lobe Abscess.)

Failure to locate the lateral ventricle by puncture would seem to indicate that if, during an explorative operation for suspected brain abscess, free incision of the dura is associated with marked herniation, and lumbar puncture fails to reveal the excessive herniation which threatens rupture of the piarachnoid, the surgeon should immediately explore the temporo-sphenoidal lobe and partially evacuate the abscess, if located. On several occasions evacuation of abscess has converted an uncontrollable cerebral herniation into an apparent collapse of the affected hemisphere, the brain no longer completely filling the skull.

*Puncture of the Opposite Ventricle for the Reduction of Brain*

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[11] Reynolds, C. E.: *Journal American Medical Association*, 1914, Vol. LXII, p. 449, contains illustration showing obliteration of ventricle and displacement of hemisphere beyond median line by a brain abscess.

*Herniation Sufficient to Admit of Elevation of Brain.* (See Lumbar and Ventricular Puncture, p. 29.) The post-mortem and clinical evidence of obliteration of the lateral ventricle with displacement of the affected hemisphere would indicate that, in the presence of marked herniation, if lumbar puncture liberates but a few drops of cerebrospinal fluid, or cerebral exploration fails to locate the abscess, puncture of the opposite lateral ventricle, through an independent opening, may reduce the intracranial pressure sufficiently to permit of inspection between the dura and the brain.

**Inspection by Elevation of Brain.**—Inspection of the under surface of the temporo-sphenoidal lobe, as far as the tegmen is desirable, because upon the presence or absence of adhesions between the dura and brain will depend the surgical procedure to be adopted.

**Technic of Elevation of Brain.**—On first opening the dura over the external surface of the temporo-sphenoidal lobe, it is generally impossible to inspect the area of limited adhesions about the middle ear by elevation of the brain, as the internal pressure of the brain is so great that it is jammed tightly against the cut edges of the dura. Although a lumbar puncture lessens the herniation, the reduction of the pressure is usually not sufficient to allow of inspection by displacement of the brain upwards. Reduction of the intracranial pressure by puncture of the opposite ventricle, or by partial evacuation of an intracerebral abscess, usually permits elevation of the brain in the anterior portion of the flap, allowing inspection of the under surface of the temporo-sphenoidal lobe above the tegmen antri and tympani—the most frequent location of intrapiarachnoid abscess and abscess of the second temporo-sphenoidal convolution "with a stalk."

By elevating the brain in the posterior portion of the flap above the horizontal portion of the sinus, the upper surface of the tegmen cerebelli, as it rises upward and inward, and the under surface of the occipital lobe, may be inspected.

Inspection of this area is difficult, as the margin of the dural opening, and the elevated postero-inferior border of the bony flap are both considerably above the outer limit of the tentorium. Introduction of the spatula between the dura and the brain is accompanied by considerable hemorrhage from injury to the posterior-inferior cerebral veins entering the sinus directly from the temporo-sphenoidal region of the brain in the triangle between the superior border of the temporal containing the superior petrosal sinus, the knee of the lateral sinus and the transverse sinus.

*Protection of Exposed Piarachnoid Prior to Exploration.*—In all cases of suspected brain abscess, before exploring the brain substance, the piarachnoid exposed by the dural opening should be covered with wet cotton to protect it from drying and from possible infection during the sudden liberation of pus through the exploring canula. The piarachnoid is a mesh of endothelial cells and fine blood vessels. It offers great resistance to trauma while moist. The brain, as long as its piarachnoid covering is intact, may be displaced and handled without injury or hemorrhage, except at the comparatively few places where blood vessels run directly from the arachnoid into the

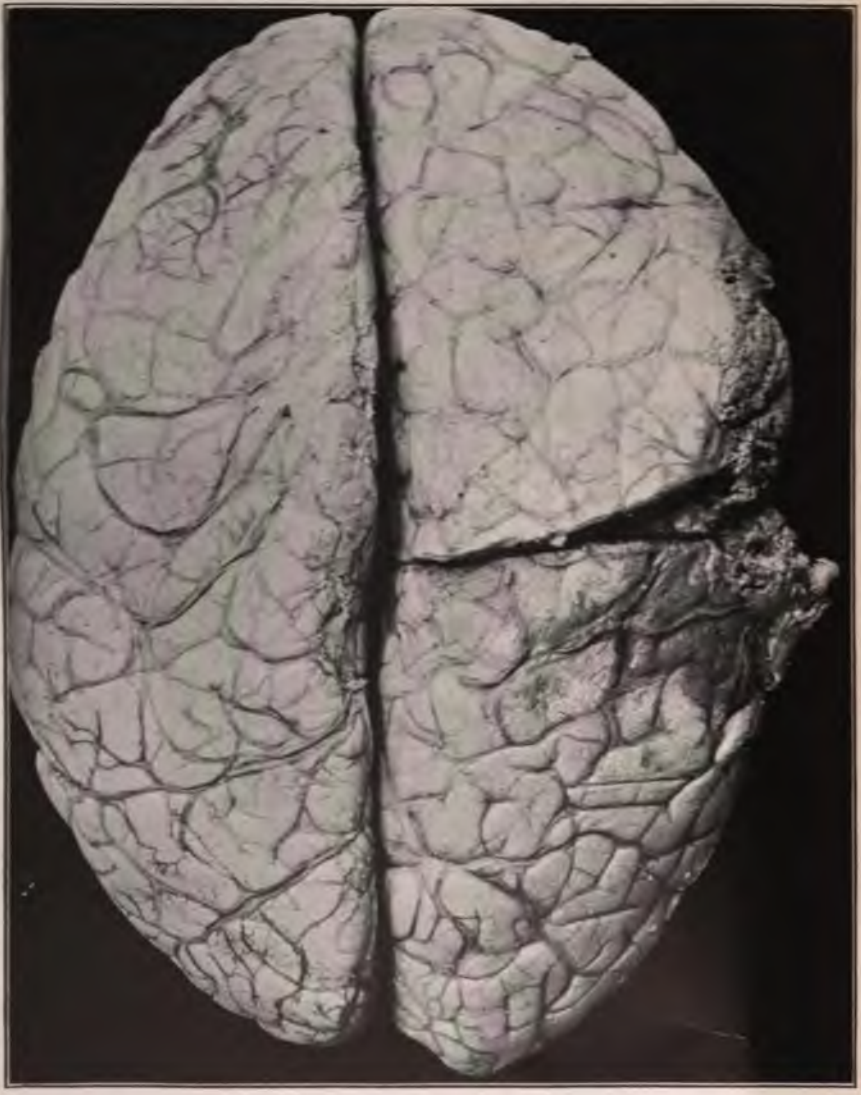

FIG. 12.—Hemorrhagic infiltration into substance of piarachnoid from tearing small pial vessels during operative manipulation. Note multiple minute hemorrhages, partially due to surgical manipulation and cerebral herniation.

dura, or outside the cranium.[12] When the piarachnoid becomes dry, however, it tears easily; even the weight of the brain, itself, may cause rupture, and, as the piarachnoid is largely made up of blood vessels, tearing is accompanied by hemorrhage. Hemorrhage into the piarachnoid is largely infiltration into the cortex itself, because of the close adhesions of the pia to the cortex. How frequently do we see the beautiful glistening surface of the cerebral cortex become dry, lustreless, and almost black with hemorrhage, as the surgeon proceeds with his operative manipulations. (See Fig. 12.)

Horsley operated within the dura under a stream of warm, weak bichloride solution. Absorbent cotton saturated with warm normal salt solution, as employed by Cushing, is much better, as it may be used to control any slight hemorrhage and, at the same time, to protect the meninges from infection.

Many cases of brain abscess, after evacuation, end fatally from meningitis, the otologist frequently giving less consideration to the protection of the piarachnoid from the pus of the abscess than the abdominal surgeon gives to the peritoneum in draining an appendicular abscess. As a matter of fact the arachnoid is much more susceptible to infection than is the peritoneum, the peritoneum passing its existence in the neighborhood of infection, whereas the arachnoid, normally, is remote from infection. (See Fig. 13, a and b.)

**Technic.**—Before searching within the brain, the piarachnoid should be covered with wet cotton, that the first gush of pus from the searcher may not soil it, after which the remainder of the pus should be removed by a suction apparatus, as it presents in the searcher.[13]

*Exploration within Cerebral Substance.*—Exploration into the brain substance should be performed with a hollow semi-blunt searcher. This is of the greatest importance. Exploration with a knife is generally associated with hemorrhage from the interior of the brain. The vessels within the cerebral substance run perpendicular to the surface; consequently a blunt searcher passing parallel to a vessel pushes it aside, whereas a knife cuts it.

The disadvantage of exploration with a semi-blunt searcher is that the capsule of the abscess may be so dense that penetration by the searcher is impossible (Fig. 18)—a very rare condition.

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[12] In exploring over the temporo-sphenoidal lobe with the spatula, after the brain has been caused to "fall back" by the withdrawal of the cerebro-spinal fluid by lumbar or ventricular puncture, one large and one or two small venous communications will be seen passing directly into the sinus. Care should be taken not to tear them before the application of "silver clips"; otherwise, hemorrhage sufficient to interfere seriously with further inspection may result.

[13] A suction apparatus with the tip at least twice the calibre of the searcher should catch the pus as it presents itself in the searcher, or in the cavity of the encephaloscope. The tip should never, of course, touch the brain tissue itself.

In Blake's case[14] it was one-sixth, and in Knapp's[15] one-fifth inch thick, while in a personal case it was so dense it could not be perforated with a sharp chisel.[16] The pus may be too thick to run through the lumen of the searcher. The contents in Bacon's case[17] had "almost the thickness of chewing gum"; in Manasse's[18] it was necessary to use a curette to remove it; while in Röpke's[19] the abscess cavity was filled with blood.

Difficulty in evacuating is encountered only in chronic abscess.

**Technic: Exploring Instrument.**—In abscess of recent formation a smooth, graduated, soft rubber catheter, lubricated, with a well fitted metal stylet, should be employed for exploration. If simple evacuation is decided upon it may, without withdrawal, be utilized as a drainage tube and for the formation of a tract. If a firm capsule is suspected, Cushing's metal searcher should be substituted, as the capsule may be impervious to the rubber searcher.

## SITE OF ADJACENT INTRACEREBRAL ABSCESS WITHOUT MACROSCOPICAL EVIDENCE OF CORTICAL INVOLVEMENT OF MIDDLE FOSSA:

Post-mortem examinations and clinical experience have demonstrated that the vast majority of adjacent intracerebral abscesses originating from the ear are situated primarily in the white substance of the third temporal convolution, directly above the tegmen tympani et antri.[20, 21] It is the involvement of this area on the left side, containing as it does the "naming" center of Mills, that gives the localizing symptoms of a pathognomonic "naming aphasia."[22]

From this original site the abscess extends either (1) inward and upward, causing a paresis, first of the lower face, then of the arm, and later of the leg; or (2) downward, towards the cortex over the tegmen.

**Selection of Site for Exploration.**—Because of the uniform appearance of the abscess in the white substance of the third temporal convolution, the site of the exploration should be as low down as possible toward the floor of the middle fossa, just above the tegmen. Otherwise the abscess, if small, may be overlooked;—this occurred to the writer in one of his earlier cases.

CASE XI., D. D.

**Abscess Low Down in Middle Fossa.** Male, 45 years of age. History; chronic alcoholism; left-sided otitis media of three months' duration; constant pain in head; vague chill for several weeks, with loss of flesh; coated tongue;

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[14] Blake: *Lancet*, I, March 31, 1900, p. 439.

[15] Knapp, H.: Archives of Otology, Vol. 29, 1900, p. 46.

[16] See Chapter Five, Metastatic Abscess, p. 68.

[17] Bacon: *Trans. Amer. Otol. Soc.*, May, 1900, p. 404.

[18] Manasse: *Arch. Otol.*, Vol. XXVII, 1898, p. 115.

[19] Röpke: *Arch. Otol.*, 1901, Vol. XXX, p. 30.

[20] MacEwen: Pyogenic Infective Diseases of the Brain and Spinal Cord; 1893, p. 72, Fig. 30.

[21] MacEwen: Atlas of the Head Sections; 1893, Plate 8.

[22] Mills' Naming Center is "located in the inferior temporal gyrus, just anterior to the middle of the inferior lateral border of the hemispheres." Santée: Anatomy of the Brain and Spinal Cord; 1915, p. 192.

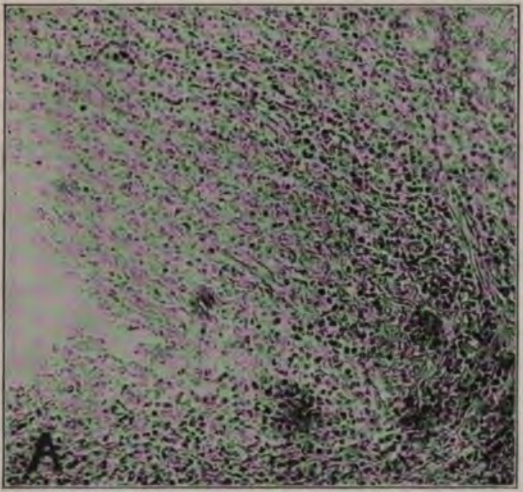

FIG. 14-A

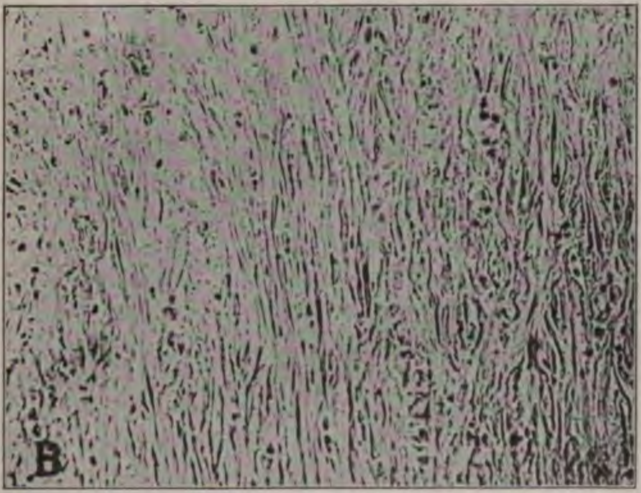

FIG. 14-B

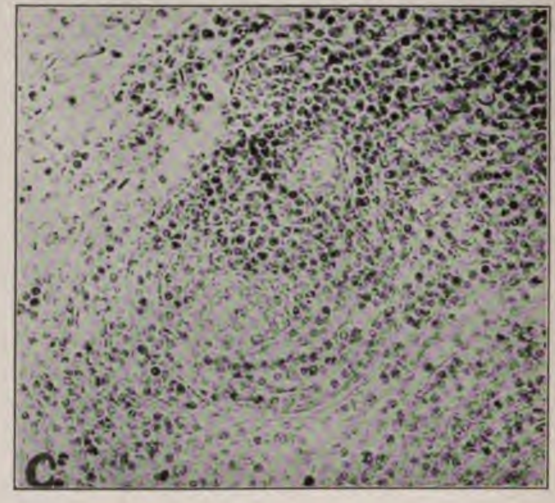

Fig. 14-C

Fig. 14 A, B, C.—Microphotograph made from sections taken from encapsulated abscess of brain of 3½ months' duration. (Dr. Harrison Martland.) See Fig. 17.

A.—Inner third of abscess capsule; showing shaggy tissue composed of necrotic, sloughing and disintegrating brain tissue containing hemorrhage, fibrin, pus (polynuclear leucocytes) and bacteria.

B.—Middle third of abscess capsule; showing fibroblastic tissue forming a dense fibrous protecting wall with scattered polynuclear and endothelial leucocytes. Fibroblastic proliferation is in this instance due to stimulation of fibroblasts by the presence of fibrin in the disintegrating brain tissue.

C.—Outer third of abscess capsule; showing endothelial leucocytes around dilated blood-vessels and in the surrounding œdematous brain tissue. Note absence of polynuclear leucocytes.

indefinite history of inability to name objects, which had entirely disappeared at time of examination.

**Mastoid Operation:** For one week apparently recovering, but headache continued. Later, attacks of transient and incomplete "naming aphasia"; slight paresis of *lower* portion of *right* face, only elicited by spontaneous emotional movements, namely, smiling or laughing.

**Second Operation:** Exploration through clean area above the ear; no abscess located. Marked improvement followed operation; patient insisted upon leaving bed; suddenly, a few days after operation fell while walking; became cyanosed; paralysis of respiration, and death.

The exploration low down toward the floor of the middle fossa has the advantage that, being adjacent to the infected mastoid area, the piarachnoid is punctured through the area of inflammatory protective changes which are always present. And puncture through a "prepared" area is much less frequently followed by leptomeningitis than through one which is "unprepared."

### CHRONIC ADJACENT INTRACEREBRAL ABSCESS WITH A CAPSULE.

**Surgical Pathology.**—The pathological processes in chronic brain abscess are the same as in abscess formation in other parts of the body, *viz.,* death of tissue and the changes due to the tissues' protective reactions, the latter being governed, however, by the special tissue involved.

Microscopically, a chronic brain abscess with a capsule is composed of: (1) A cavity filled with detritus and pus (Fig. 14A) the end result of complete death of tissue from bacterial action and nutritional disturbances, and (2) a limiting capsule, formed by the irritation of the bacteria and their toxins on the tissues, and the tissues' protective and reconstructive reactions. The capsule in a general way may be divided into three zones, all closely intermingled, the destructive bacterial action having predominance nearest the cavity, while in the external zones the tissues' reconstructive and protective powers are chiefly manifest; although far beyond the capsule, bacterial action can always be demonstrated.

Just external to the cavity and projecting into it is (1) the "necrobiotic zone," in which the tissue now dead is undergoing liquefaction from the action of the toxins. To insure the ideal surgical result, namely, the eradication of the infection, the surgeon must remove this zone, as far as possible, using the utmost delicacy. Merging into the necrobiotic zone is (2) the granulation tissue zone, with connective tissue developing from it—the capsule (Fig. 14B); and external to both in the otherwise normal brain tissue is (3) a round-cell infiltration in the perivascular sheath of the vessels, showing that the inflammatory or the protective process is still active well beyond the capsule. (See Figs. 14C, 15 and 16.)

The capsule of connective tissue is the result of two factors—the resistance of the tissues and the virulence of the infection. With a high resistance and a low virulence, nature attempts a complete encapsulation of the abscess by a wall of new connective tissue, which grows denser with time.

The connective tissue reaction of the body is simply the attempt of the organism to limit the extent of the inflammatory process. In most parts of the body, subcutaneous, muscular, etc., if the infection is not virulent, an encapsulation rapidly follows, partly because the tissues in general are freely supplied with connective tissue which, being of a low order of cell, rapidly propagates itself. In the brain, however, the formation of a connective tissue capsule is a considerably slower process; partially because practically all the new cells making up the capsule must be brought to it by the newly formed blood vessels. These blood vessels, consisting at first of only an endothelial tube, soon develop an outer connective tissue layer, and from this layer is formed the connective tissue of the capsule. (See Fig. 14B.) The connective tissue of the piarachnoid also plays a part, but the glial cells, the framework of the brain, being highly specialized connective tissue, plays little or no part in the encapsulation.

In the formation of a capsule the type of micro-organism is thought by some to play a large part, Neumann [23] stating that "diplococci cause an abundant secretion of fibrin, and so form a capsule, but when anerobic micro-organisms are present, a firm capsule is not formed."

While, microscopically, the formation of connective tissue can be demonstrated within a short period, the development of a capsule firm enough to be appreciable macroscopically and consequently of great surgical assistance, is a slow process. Friedmann [24] found microscopically a distinct membrane, composed of two layers, between the eighth and the twelfth day after the cerebral infection. The resistance to the advance of the searcher offered by such a capsule would not be appreciable to the surgeon. Westphal [25] found a delicate encapsulating membrane seventeen days after the onset of the cerebral symptoms. In such a case it is probable that, with very delicate handling of the searcher the surgeon would be aware

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[23] Neumann, H.: Cerebellar Abscess, English Ed., 1907, p. 14; Politzer: A Textbook of Diseases of the Ear, 5th Ed., 1909, p. 66.

[24] Friedmann: Handbuch der Pathologischen Anatomie des Nervensystems, 1905.

[25] Westphal: *Archiv für Psychiatrie,* 1900, Bd. 33, S. 206.

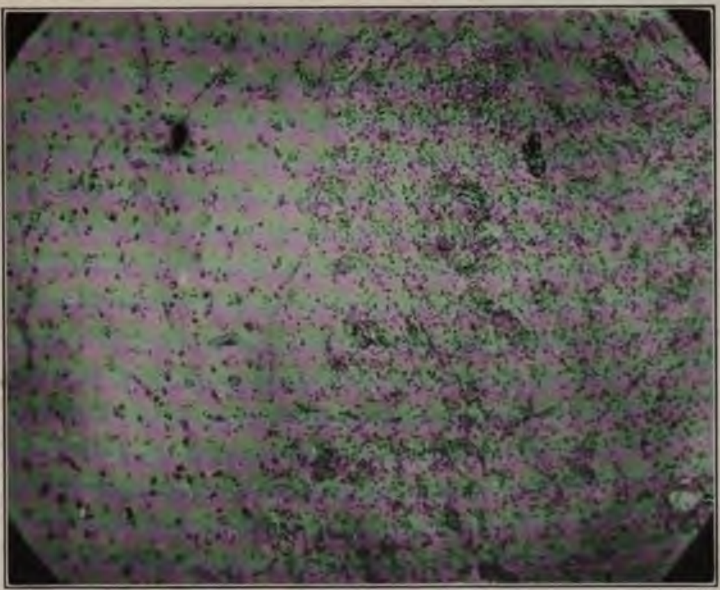

FIG. 15.—Section (Dr. Fredk. Sutton); low power: through granulation layer of chronic brain abscess of about 14 days' duration. To right, new connective tissue: perivascular infiltration. To left, normal brain tissue.

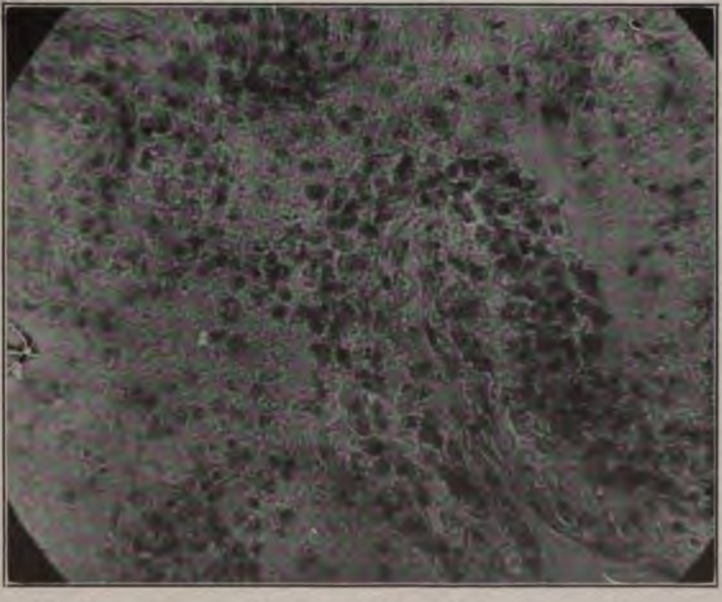

FIG. 16.—Same case as Fig. 15. Section; high power: perivascular infiltration.

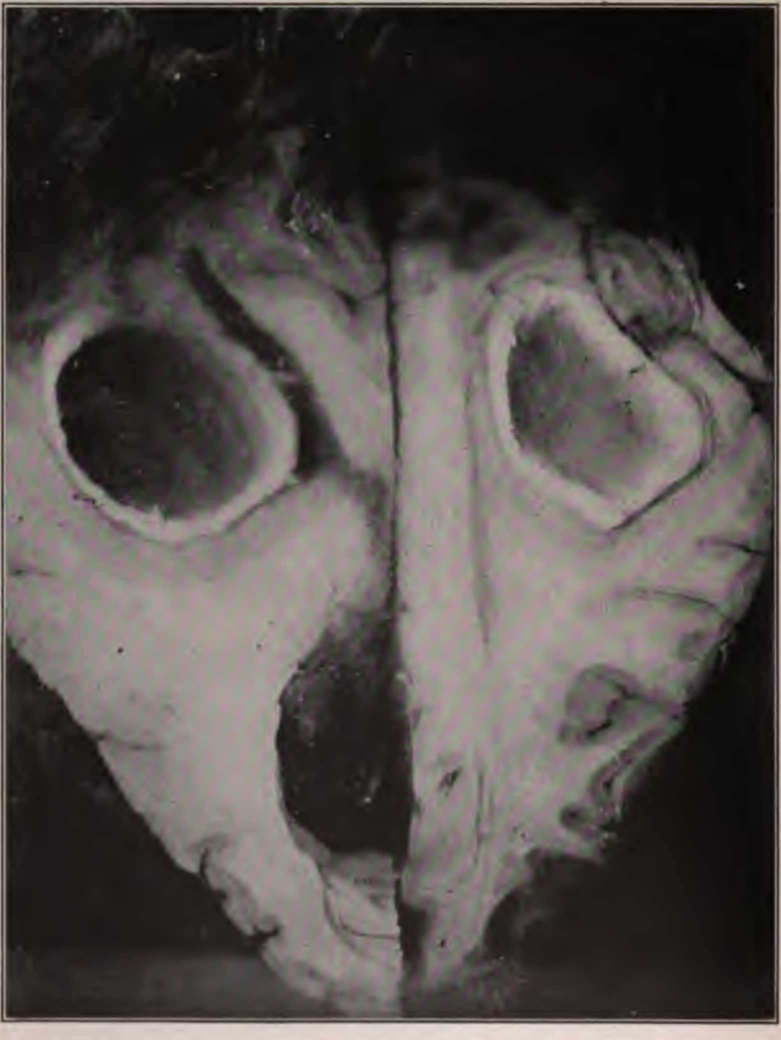

FIG. 17.—Section through streptococcic brain abscess of 105 days' duration. Firm capsule ¼ to ⅓ inch in thickness. (For microphotographs of sections see Fig. 14.)

of a slight resistance at the moment of perforation. Uchermann[26] estimated that it probably takes about 24 days to form a capsule of one millimeter thickness; about 38 days for one of two millimeters. The latter condition would offer considerable resistance to a semi-blunt searcher and would be impervious to a blunt instrument. (Fig. 17.)

Although several clinical observers[27, 28, 29] contend that a capsule is not formed until between the third and eighth week, the author has recognized the presence of a capsule as early as the seventeenth day.[30] After the incision of the piarachnoid the consistency of the brain tissue is so uniform that, by delicate handling, very slight resistance is appreciable.

## SURGICAL PRINCIPLES UNDERLYING THE INTRADURAL TECHNIC IN SUSPECTED, ADJACENT, (SECONDARY) ABSCESS OF THE MIDDLE FOSSA (TEMPORO-SPHENOIDAL).

As the diagnosis of brain abscess must of necessity be more or less provisional, and as cortical and subcortical suppuration can rarely be differentiated, all explorations must be conducted through a clean area, and adequate exposure must be provided for.

In no field of surgery is it more important that the surgeon keep an open mind. Because of the uncertainties of the diagnosis, he must prepare to assume a "shifting policy," depending upon whether the exploration reveals, in chronic abscess, an intracerebral abscess without macroscopical evidence of cortical involvement, with a stalk, or meningeal; and in acute abscess, an intracerebral or meningeal abscess.

He must not adhere to a particular procedure simply because it resulted successfully in a former operation. He must adapt each act of his technic to the condition found, preserving the protective structures erected by nature, and availing himself of their assistance. Compression, because it favors extension of suppuration and paralyzes the protective mechanism, must be controlled or prevented.

In the treatment of individual clinical types of abscess (see p. 21), existing pathological conditions and clinical experience both suggest that the surgeon follow the method adopted by nature in its efforts to cure spontaneously an adjacent brain abscess.

In encapsulated adjacent abscess with a stalk, as nature has erected a walled-off tract through the piarachnoid into the cavity of the abscess, the surgeon should utilize this tract for the complete evacuation, cleansing, and obliteration of the abscess and the introduction of necessary drainage. In adjacent intracerebral abscess with capsule, but without macroscopical evidence of cortical involvement, the abscess should be completely evacuated and an attempt made to form a walled-off area through the piarachnoid, after which it may be completely evacuated, cleansed and obliterated. In acute abscess, as compression is favoring the extension and preventing the limitation of the suppuration—as well as being the cause generally of most of the clinical symptoms—the abscess should be evacuated as completely as possible through a small searcher and the dura closed, or at least the brain completely covered. In several instances I have firmly anchored the small searcher to facilitate the formation of a tract. Later, after the formation of the capsule, it may be treated as an encapsulated intracerebral abscess. A meningeal abscess, chronic or acute, should be fully exposed in all directions, no matter how extensive the sacrifice of bone or dura, the area should be thoroughly cleansed by irrigation, adequate drainage provided at a distance from the dural margin through "stab" wounds, and the brain completely covered.

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[26] Uchermann: *Zeitschrift für Ohrenheilkunde*, 1904, Bd. 46, S. 306.
[27] Lebert: *Archiv für Pathol. Anat. und Physiol.*, 1856, Bd. 10, S. 78.
[28] Schott: *Würzburg. Med. Zeitschrift*, 1861, Bd. 2, S. 462.
[29] Huguenin: *Handbuch der spez. Pathol. und Therap.*, 1876, Bd. 2, S. 331.
[30] See further discussion of formation of capsule, Chapter Five, Metastatic Abscess, p. 76.

**Technic Following Elevation of Brain.**—A lumbar or ventricular puncture of the dilated opposite ventricle, or the partial evacuation of the abscess, through the cortex, having caused sufficient "falling back of the brain" to allow an intradural inspection over the roof of the tegmen if adhesions are seen above the middle ear, all of the exposed external surface of the temporo-sphenoidal lobe should be covered with wet cotton, the bone rongeured away down to and including the middle ear and antrum, and the dura opened from below.

If the stalk of the abscess is short so that the abscess cavity is separated by only a few millimeters from the under surface of the temporo-sphenoidal lobe, it may be possible to eradicate it, viz., to evacuate, cleanse, obliterate and introduce drainage material through the lower dural opening, in which case the upper dural opening should be tightly closed and the bone flap replaced.

In cases in which the protective inflammation is very limited, this area may be increased by placing strips of moist cotton, from the upper wound, between the dura and the brain on as many sides of the adhesive area as possible, before opening from below.

### SURGICAL SIGNIFICANCE OF AREAS OF ADHESIVE INFLAMMATION OF PIARACHNOID TO DURA.

When elevation of the brain with the spatula discloses, on the under surface of the temporo-sphenoidal lobe, adhesive inflammation uniting the brain with the dura, it may be inferred that if the area of adhesion is extensive, the abscess is probably in the meshes of the piarachnoid; and that if the adhesions are limited to the tegmen, the abscess is probably in the brain substance, a "stalk" uniting the abscess with the area of primary infection.

Both forms of abscess offer excellent prospects of recovery, for nature has walled off the infected tract from the meshes of the piarachnoid, and established a tract from the dura to the furthest extremity of the abscess, while the adjacent tissues have become largely immunized during the limitation of the infection.

## CHRONIC ADJACENT INTRACEREBRAL ABSCESS OF THE MIDDLE FOSSA.

It is recognized that a larger proportion of recoveries have followed evacuation and drainage from below through the middle ear and antrum than through the external surface of the temporo-sphenoidal lobe, because in abscess with a "stalk" the tegmen permits entrance into the abscess at its nearest approach to the dura, and through an immunized tract of protective adhesions. Consequently, in operating, a definite attempt should be made to evacuate the abscess at its nearest approach to the dura,—the antrum and middle ear,—and to utilize the area of protective adhesions,—for it is through this tract that the abscess may be entered, thoroughly evacuated, and drainage material introduced with the least danger of inciting a suppurative meningitis, or of injuring and infecting the neighboring cerebral tissue, with its accompanying œdema, encephalitis and fungating hernia.

### Surgical Principles and Specific Factors Involved in the Treatment of Encapsulated Adjacent Intracerebral Abscess of the Middle Fossa.

In the surgical treatment of encapsulated intracerebral abscess one of two principles is employed: (1) Attempted eradication of the suppuration, and (2) drainage.

Drainage, while efficiently controlling suppuration in other parts of the body, is generally inadequate in cerebral suppuration (see p. 57), and may be the direct cause of secondary infection of the meninges, the development of the so-called secondary abscess, and of hernia cerebri.

On the other hand, an attempt at eradication, except in selected cases, will be associated with cerebral trauma, resulting in the presence of infection in traumatic encephalitis.

On the proper selection of the principle to be adopted in each individual case, largely depends the life of the patient.

**Eradication.**—In the treatment of chronic intracerebral abscess with a capsule with a stalk, the surgical aims should be the same as in the surgical treatment of low grade infection in other soft tissues, *viz.:* (1) thoroughly to remove all pus and detritus, thus largely eliminating the infection; (2) to reduce as far as possible the suppurative surface by the *collapse* and *obliteration* of the cavity; (3) to convert the granulating surface of the obliterated cavity into an active reparative process, the subsequent discharge being then largely the discarded products of healing, and not, as formerly, the result of the destruction of tissue. In addition to this the surgeon must endeavor, (4) to accomplish the above without spreading the infection to the susceptible adjacent piarachnoid; and (5) to do so with a minimum of operative trauma.

**Drainage.**—In encapsulated intracerebral abscess without macroscopical evidence of cortical involvement, complete evacuation, cleansing, and obliteration are impossible, because wide entrance into the abscess is apt to be followed by meningitis (there being no area of protective adhesion) and traumatic encephalitis from injury to cerebral tissue.

**Selection of Cases.**—When the falling back of the brain reveals a wide stalk leading into an abscess cavity, an attempt should be made at complete evacuation, cleansing, and obliteration of the cavity. Again, if exploration of the under or lateral surface of the temporo-sphenoidal lobe reveals an encapsulated intracerebral abscess without macroscopical evidence of cortical involvement, within one or at most two centimeters of the cortex, attempted eradication is justifiable, although without a stalk a systematic effort at complete evacuation, cleansing and obliteration, increases the danger from meningitis and traumatic suppuration.

With an intracerebral abscess without a stalk, situated beyond two centimeters from the cortex, an attempt at eradication is associated with great technical difficulties and prohibitory trauma to the brain. The surgical effort should be limited to as complete an evacuation as is possible through the searcher, and the formation of a tract through which the abscess is drained and later, possibly, cleansed.

**Difficulties of Complete Evacuation.**—Certain anatomical and physiological factors peculiar to cerebral tissue make difficult the complete evacuation of the pus and obliteration of the cavity:—(1) the brain substance has a specific gravity of 1036,[31] 78.9 per cent. of it being water; (2) the brain substance is practically a semisolid, homogeneous mass of nerve cells and glial tissue, while the blood vessels within the substance are comparatively small;[32] (3) the brain is largely held in shape by the enveloping piarachnoid (witness its distortion upon removing the fresh brain from the calvarium and stripping off the piarachnoid).

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[31] Santee, H. E., Anatomy of the Brain and Spinal Cord, 1915, p. 49.
[32] Mallory, Principles of Pathology and Histology, 1914, p. 625.

The mechanical factors inherent in the abscess itself which influence the operative procedure and modify the surgical aims, are: (1) the thickness of the enveloping capsule; (2) the distance of its outer wall from the point of puncture on the cortex; and (3) the consistency of the pus.

Taken in its entirety, cerebral substance within the piarachnoid is approximately of the consistency of mush contained within a delicate gauze bag. If now a raw egg with the shell removed is placed within the mass and an effort is made to remove the entire yolk with little disturbance of the mush, we have a fair model of the mechanical difficulties of thoroughly evacuating an intracerebral abscess with a delicate encapsulation. Puncture through the mush into the egg will cause the liberation of some of the yolk, but collapse of the surrounding mush will uniformly prevent the evacuation of the larger part of the semi-liquid contents of the enveloping membrane.

Upon entering an intracerebral abscess with the searcher, sudden expulsion of a portion of the pus is caused by the tension of the surrounding cerebral substance; but as soon as the tension between the abscess contents and the surrounding cerebral tissue is equalized, further evacuation of the pus is accomplished by gravity, and the weight of the surrounding cerebral tissue. Muscular contraction of surrounding tissue—so powerful a factor in the evacuation of the pus of an abscess in the soft structures—plays no part.

The nearer the pus approaches the density of the surrounding cerebral tissue, the greater the mechanical difficulty of complete evacuation. Pus of thin consistency will be much more completely evacuated by the brain than thicker pus.

The firmer the envelope, the greater the possibility of entrance into the abscess cavity of an instrument which will insure complete evacuation, cleansing, and obliteration of the cavity.

**Importance of Thorough Primary Evacuation.**—Complete evacuation, cleansing and obliteration of the cavity whenever feasible without trauma to normal cerebral tissue, should be carried out *at the time of the primary operation.* In recent years I have realized more and more that, having once located the collection of pus with the exploring searcher, thorough evacuation and obliteration of the cavity greatly lessens the liability of subsequent re-filling, or secondary abscess from inadequate drainage.

The literature of the treatment of chronic brain abscess shows how frequently reliance is placed on partial evacuation, with the introduction into the cavity of some form of drainage material to allow of subsequent drainage of pus. I am convinced, however, that not only is incomplete primary evacuation, preventing obliteration of the cavity, generally followed by inadequate drainage (because of the collapse of the opposing walls shutting off portions of the abscess with the frequent formation of an apparently new cavity), but that the retained pus favors an extension of the encephalitis. Many failures to locate pus at a *secondary* operation, in which primary incomplete evacuation had failed to stop the suppurative process, and in which at post-mortem the drainage material was found outside the abscess cavity, have persuaded me that the surgeon, in chronic brain abscess with a capsule, has but one major chance to combat infection, namely, *at the time of the primary operation.*

The importance of primary complete evacuation is supported by two clinical observations of great prognostic value, which have long been recognized: (1) if following an operation for brain abscess, the disappearance of the symptoms is *accompanied by almost complete cessation of the discharge,*—complete primary evacution—relapse and refilling of the abscess cavity but rarely occurs; (2) on the other hand, continuation after the first few days of a profuse discharge from the drainage tube—incomplete primary evacuation—frequently results in recurrence of the symptoms associated with a sudden cessation of the discharge, cleansing of the tube showing that the cessation of the discharge is not due to "plugging" of the tube; the new area of encephalitis being apparently not connected with the region drained.

Evacuation through puncture of the dura is recommended by Lemaître.[33] This gives a large percentage of recoveries because it evacuates the abscess with but a slight amount of trauma. However, it has the great disadvantage that a subtemporal and piarachnoid abscess either will be entirely overlooked or imperfectly evacuated.

The method fulfills the surgical requirement for the successful treatment of acute brain abscess; it results in evacuation and the formation of a tract. However, before the method is employed care should be taken to be assured that the abscess is not chronic, as otherwise the operation will fail. In several of the writer's cases with an abscess with a capsule which approached the surface it was evacuated in this way, mistaking it for an acute abscess. Post-mortem revealed that, had the abscess been properly eradicated, the patient undoubtedly would have recovered.

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[33] Lemaître, F.: Exclusion of Subarachnoid Space; *Revue de Chirurgie*, 1919, Tome 57, p. 497. (Abstracted in *Jour. Am. Med. Soc.*, June 12, 1920, Vol. 74, No. 24, p. 1680.)

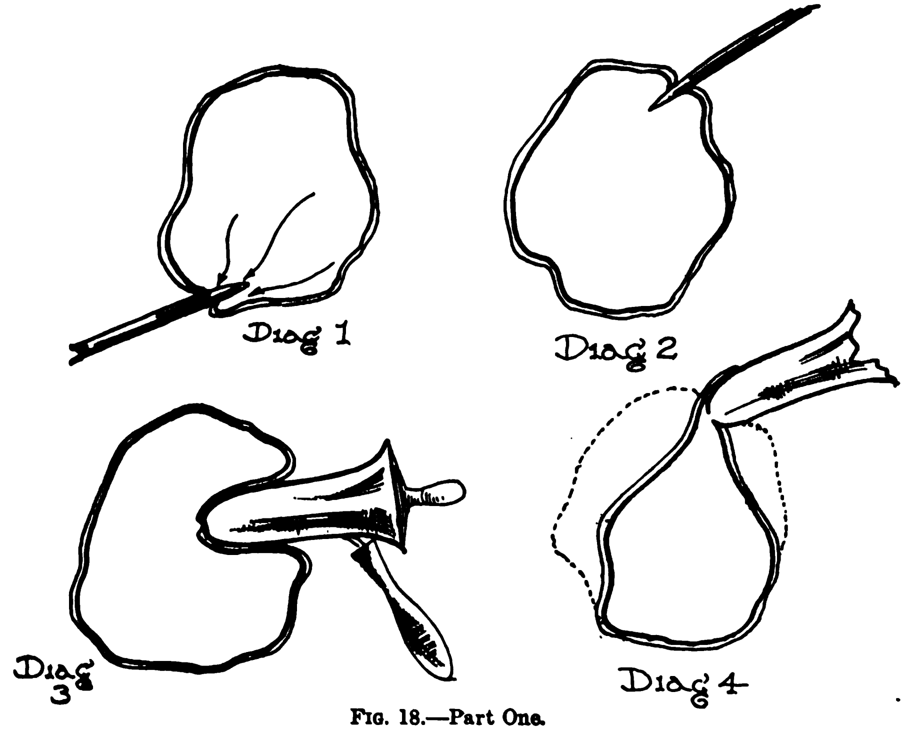

Fig. 18.—Part One.

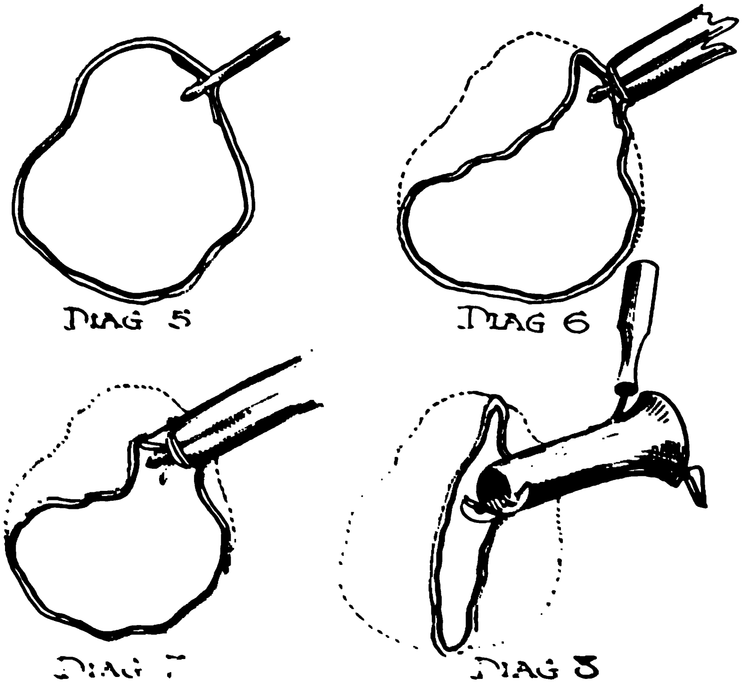

Fig. 18. Part Two.
Collapse of abscess, 4, 6, 7, 8. Difficulty in entrance by blunt instrument, 3, 4, 6. Capsule holders in place, 5, 6, 7, 8.

### TECHNIC OF SURGICAL TREATMENT OF CHRONIC ADJACENT INTRACEREBRAL ABSCESS WITH CAPSULE.

**Complete Evacuation; Difficulties.**—The technic depends upon whether eradication—complete evacuation, cleansing and obliteration of the cavity—or simple drainage is to be attempted. In evacuating a chronic intracerebral abscess with a limiting membrane situated fairly near the surface, after the first rush of pus has been removed care should be taken to evacuate only a small quantity of pus through the exploring searcher. Unless this precaution is taken the exploring searcher, having entered a small abscess or perforated a dense capsule, the partial withdrawal of the pus will result in the collapse of the opposing cavity walls from the external pressure of the surrounding brain tissue, thus locking in a quantity of pus and rendering difficult the proper placing of the encephaloscope. The more pus evacuated the greater the flaccidity of the portion of the abscess wall impinged upon by the blunt encephaloscope; consequently, by preserving the distention of the abscess the difficulty of perforating the capsule by the encephaloscope and placing the instrument within the abscess cavity is lessened. The fact that the encephaloscope is correctly placed is shown by the free escape of pus through the lumen.

This locking in of pus invariably happens in all intracerebral abscesses, chronic or acute, when evacuation is attempted through the exploring searcher, unless the searcher has entered the abscess cavity at its most dependent portion and has passed well into the cavity itself. If the searcher has entered the upper portion of the abscess (especially if the abscess is multilocular in shape), the partial evacuation of the abscess with the collapse of its walls, will leave the remainder of the pus at such an angle from the exploring searcher's tract, that if it is necessary that another instrument, such as an encephaloscope or forceps carrying drainage material be passed along the searcher it may not enter the cavity, this portion of the cavity having now become a mere slit between two opposing walls and away from the remainder of the still pus filled cavity, to which it is united by an obliterated tract at right angles to the exploring searcher. This tract can be followed if the abscess is held by the retaining searcher, by lifting apart its collapsed walls, thus allowing entrance into all parts of the remaining cavity. (Fig. 18.)

The writer has frequently failed to find the cavity of the abscess after the evacuation of a quantity of pus through the exploring instrument, although considerable pus was still locked in an extremity, the position of the remaining pus being distant perhaps by several centimeters from that portion of the cavity obliterated by the partial evacuation.

*An abscess once entered by an exploring instrument should never be lost.* This is of the utmost importance. As soon as pus is definitely located by the searcher its flow should be stopped, because when the abscess becomes flaccid greater difficulty is experienced in finding it with the abscess holder, and in entering it with the inspecting instrument. In the case of a chronic intracerebral abscess with a firm capsule, if the capsule is of delicate structure, on passing the encephaloscope over it pus will appear in the instrument at the recorded depth. If, however, the limiting membrane is of more than three weeks' duration, it may be dense enough to resist the entrance of the encephaloscope, in which case, although the cavity of the encephaloscope remains free from pus, the pressure will appreciably increase the flow of pus through the exploring canula. The abscess is again perforated by the abscess holder and if on withdrawing the stylet pus flows from it the inspecting instrument is passed over it.

Although Whiting's encephaloscope[34] is invaluable for the complete evacuation, cleansing and obliteration of the cavity and for the introduction of drainage material, on many occasions the writer has been unable to place it properly within the abscess cavity because, (1) it is introduced *beside* the searcher and, consequently, if the searcher has perforated an extremity of the abscess (Fig. 18, Diagram 3), the encephaloscope may miss it entirely; and, (2) because, being blunt, the instrument may simply indent a firm abscess capsule (Fig. 18, Diagram 4). Both accidents have occurred in the writer's experience.

To obviate the former accident the author has devised a modification of Whiting's encephaloscope, which is to be introduced over the exploring searcher, whose track occupies the center of the inspecting instrument. (Figs. 19, 20, 21.)

### CLEANSING AND OBLITERATION OF CAVITY OF ABSCESS WITH "STALK" OR NEAR CORTICAL SURFACE.

A brain abscess with a capsule, if near the cortical surface, should be thoroughly evacuated, its walls inspected and cleansed, and its cavity obliterated, so that on withdrawal of the inspecting encephaloscope, the surgeon should *know* that no recess of the abscess has been overlooked. Examination of recorded cases of "second" brain abscess demonstrates that separate and distinct multiple brain abscesses (excepting those of metastatic or traumatic origin) are very rare—"second abscess" simply being a prolongation of the original

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[34] Whiting, F.: The Differential Diagnosis of Acute and Chronic Brain Abscess by Means of the Encephaloscope. *Transactions American Otological Society*, 1903, Vol. VIII, p. 225.

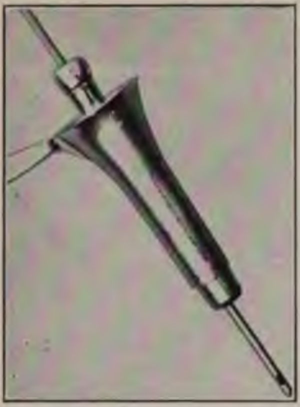

FIG. 19.—Author's modification of Whiting's encephaloscope. Allows of passage of encephaloscope over searcher; aimed to prevent the "losing" of the abscess after its partial evacuation —a frequent and disastrous accident. This instrument is adapted only to cases which, from their comparatively short duration, are believed to be surrounded by a delicate capsule.

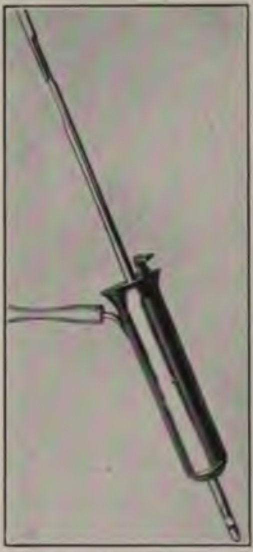

FIG. 20. — Author's capsule retaining searcher and encephaloscope (closed).

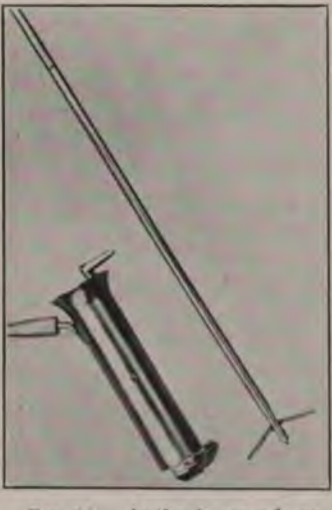

FIG. 21.—Author's capsule retaining searcher and encephaloscope (open). Adapted only to cases surrounded by a very firm capsule which will allow of manipulation and traction outward from the interior of the capsule.

abscess united to the main cavity by a narrow entrance which has been obliterated by the collapse of the surrounding cerebral tissue during evacuation.

**Technic.**—Complete illumination of all hidden recesses is of primary importance; a Klaar's headlight worn by the operator has proved most satisfactory.

All instruments used within the brain should be held by the operator himself, whose eyes should never for an instant leave the encephaloscope, the obturator or a cleansing instrument being placed in his hands by an assistant. The re-introduction of an obturator, with the slightest forward or lateral movement of the instrument, will prevent tearing and consequent confusing hemorrhage. In the latter part of the thorough evacuation, cleansing by absorbent cotton on applicators and moistened with salt solution, furnishes the greatest assistance. If the abscess is superficial, situated within two centimeters of the surface, the entire cavity may be cleansed by this method.

**Drainage.**—In a chronic abscess, with a firm limiting membrane, some form of drainage is desirable, although in my experience the subsequent discharge in carefully cleansed abscess has been so slight that with our present understanding of tissue disinfection and control of localized infection by the tissues themselves, many cases of chronic brain abscess do not require drainage. Personally, however, the writer never has had the courage to omit drainage. If the evacuation has been thorough, the very slight subsequent discharge is simply the product of tissue repair; "free drainage," in the sense of combating infection, plays no further part.

**Technic.**—Before withdrawing the encephaloscope, a wick of rubber tissue should be placed within the cavity. Gauze in any form is very irritating, as is shown by the invariable hemorrhage which accompanies its removal. The irritation of its presence favors the development of hernia cerebri.

**Technic of Drainage Following Incomplete Evacuation.**—If the external wall of the abscess cavity is more than two centimeters from the surface of the cerebral cortex, the abscess is to be as completely evacuated as is possible through the exploring instrument, assisted by gravity.

After the stoppage of the flow of pus, turning the patient in different directions, to the right or left, lowering or elevating the head, may liberate additional pus.

Because of collapse of the abscess walls and the attending difficulty of introducing drainage material within the cavity (see p. 55), the exploring instrument (graduated soft rubber catheter) should be utilized for subsequent drainage and the formation of a tract, and firmly anchored to the soft parts.

In one instance, in a deeply situated abscess, as it was impossible to enter the abscess cavity with an investigating instrument, the writer used the metal searcher as a permanent drain by cutting it off a little beyond the cortical surface. Rubber drainage tubing is much better, as later, if the abscess is deeply situated, and if its limiting membrane is firm enough to have offered considerable resistance to the passage of the searcher, cleansing by irrigation through the searcher offers the best prospect of cure. The filling of the abscess cavity is accomplished by the weight of the syringe piston, and it is emptied by means of suction.[illegible]

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[illegible] Muck has demonstrated clinically that during the dressing of a brain abscess the rotation of the head in the sitting position markedly influences the size of the

**Secondary Compression.**—Compression favors suppuration and prevents the protective and nutritive mechanisms of the brain from functioning. With a closed dura, excessive action of any of the processes—infective, traumatic, or reparative—may cause increase of the intracerebral pressure, the compression being followed by extension of any remaining infection; consequently, after evacuation, the decision must be made whether the dura shall be more or less tightly closed or whether a subsequent enlargement of the brain's bulk demands a large dural opening.

In deciding whether the dura is to be closed, the most important consideration is: Has the suppuration been completely controlled, and if so, will the secondary reactive œdema be excessive?

Without complete primary evacuation, there will be extension of the suppuration from refilling of the abscess or its extension into the already infected adjacent area, thus causing compression.

With compression from excessive secondary œdema, especially when associated with injury to normal cerebral tissue such as may accompany attempts at eradication, the remaining slight infection is given a new start. Even with complete evacuation the normal reactive forces of the brain are to be considered, for if excessive they may give rise to compression.

In brain abscess, especially acute abscess, the nutritive and protective mechanisms of the brain are greatly disturbed by the infection itself, by limitation, and by operative trauma. Following evacuation these mechanisms, in their efforts at re-establishment, may occasion such excessive re-active changes as to give rise to compression. (Fig. 22.)

In a review of the author's cases he is convinced that three resulted fatally from subsequent compression—probably preventible—which caused extension of the suppurative process (Case VIII, A. M. N., Chapter Four, p. 37, and Case IX, G. W., Chapter Four, p. 38); while in at least two others (Case XIII, Mrs. H., Chapter Four, p. 63, and Case XVII, Miss M. D., Chapter Five, p. 77), the large dural opening, one resulting in a large hernia cerebri, probably became the saving factor. (See also Case X, C. H., Chapter Four, p. 43.)

**Principles in Technic of Decompression.**—To leave the dura open is to risk a most distressing hernia cerebri, with its attending

abscess cavity and consequently its drainage. The size of the cavity is altered, he believes, by the hindering of the escape of the venous blood from the head through pressure induced by rotation of the head.

Muck, C.: Wie soll der Hirnabszesskranke nach der Operation gelagert werden? (Position of Patient after Operation for Brain Abscess); *Zeitschrift für Ohrenheilkunde*, 1920, Vol. 79, p. 86.

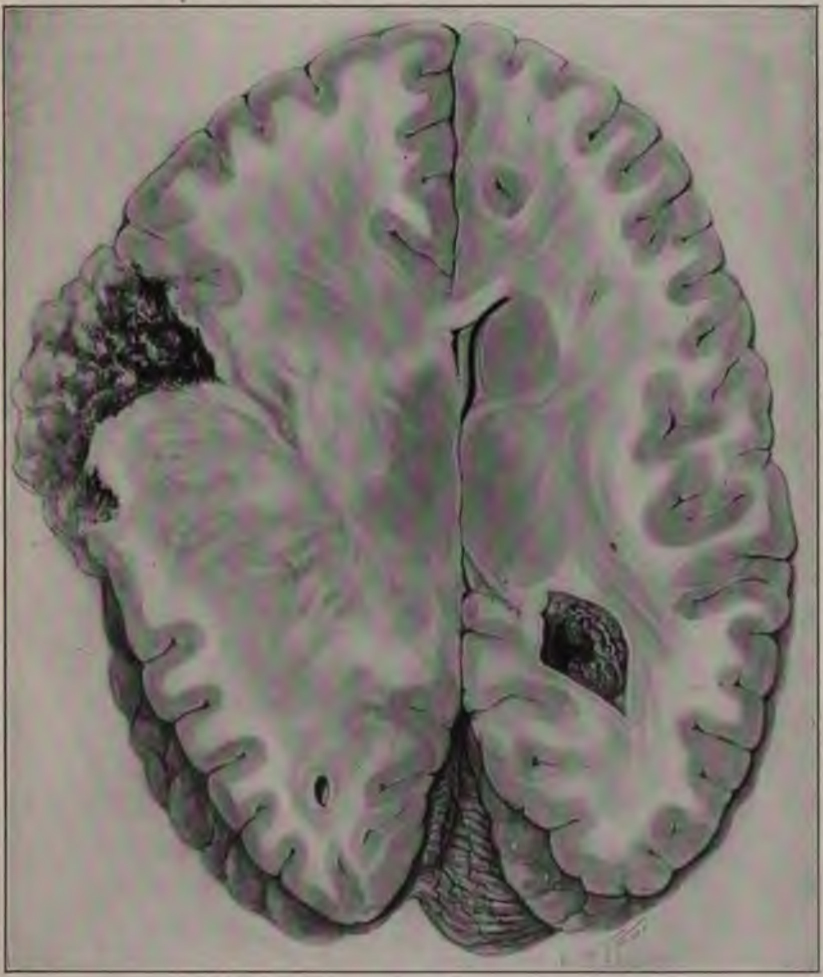

FIG. 22.—Original drawing from brain containing a temporo-sphenoidal lobe abscess which had been almost completely evacuated. Shows obliteration of the posterior horn of ventricle on abscess side and the dilatation of the ventricle of the opposite side; shows, also, extension of infiltration and increase in size of hemisphere.

danger of secondary infection, and yet not to provide for compression may be fatal. Decompression, by displacement of the brain substance, is in itself a cause of secondary œdema. If the dura is to be left open a wide opening must be provided; otherwise the herniation through a small opening may cause strangulation of the brain. With a decompression the brain must be entirely covered by soft parts.

The sewing in of a large piece of fascia lata furnishes a most efficient protection for the brain in non-suppurative cases. Its similarity to the dura renders it less irritating to the cortex than either muscle or skin.

**Immediate After-treatment.**—Following operation the patient should *remain on the operating table for many hours,* preferably until the next morning, or at least until complete consciousness has been regained. To move a patient immediately after a profound operative disturbance of his cerebral tissue adds an unnecessary tax to his endurance which may result disastrously. Since attention has been called to this important detail by Cushing,[36] the writer has been impressed by the frequent collapse of patients after being transported to the ward, not only in cerebral cases but in other types of serious operation.

If the hospital routine cannot permit the brain abscess cases to remain in the position operated upon, the institution is not a proper place for the operation.

**Continued After-treatment.**—After thorough evacuation, most delicate cleansing, and complete obliteration of the cavity, the dura and the external wound should be almost entirely closed, and no matter how great the temptation to do so, the brain itself should not again be disturbed for many days; if possible, not for several weeks. In the author's experience further manipulation invariably militates against the patient's recovery. The drain may be allowed to be gradually expelled from the brain itself. In most of the successful cases re-introduction of drainage has been unnecessary.

### "SECOND ABSCESS," OR DOUBLE AND SECONDARY ABSCESS.

In traumatic brain abscess (secondary infection of necrotic cerebral tissue, blood detritus, etc.), two or more separate abscesses frequently occur.

In abscess from direct extension from adjacent suppurative lesions or from retrograde thrombophlebitis with nutritional death, two or three abscesses must of necessity be of infrequent occurrence. Most of the abscesses recorded as "second," or "double," are in reality secondary, though a few cases of double adjacent abscess are reported.[37] A second abscess, originating from the original brain abscess is, however, of frequent occurrence, but from an etiological standpoint such are rather secondary to the original adjacent abscess and should be so designated.

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[36] Cushing, Harvey: Tumors Nervus Acusticus; 1917, *W. B. Saunders Company,* Philadelphia and London.

Secondary abscess is due, in order of frequency, to (1) "loss of abscess" before complete evacuation, or by collapse of its walls; (2) encephalitis induced in surrounding tissue by the trauma of exploration or evacuation; (3) encephalitis by direct extension from the primary abscess, the path of infection, though frequently small, being discoverable by careful examination.

CASE XII.

W. K.: *Double Cerebellar Abscess, the second abscess resulting from loss of primary abscess associated with cerebral trauma;* operations; recovery.

Boy, aged 14, always in good health, but had poor memory. Six years ago had discharge of pus from right ear for three weeks. Mother said at times a whitish mass protruded from the ear, which was very sensitive to touch.

**History.**—Awakened in the night with severe pain which lasted three days. He was then struck on the head by a ball, knocked down, but not rendered unconscious. Two days later the ear pain returned, followed in three more days by a flow of foul pus from the ear, which lasted a week. During this week he suffered from pain in the head and ear, occasional vomiting, slight attacks of screaming without apparent cause; probably dizzy, as he would catch at articles in going about the room. Lost flesh rapidly. Indistinct history of a chill. At the end of seven days he became drowsy and in the next three days seemed dazed and yawned frequently. On the eighteenth day he was found lying on his right side on the floor, his eyes wide open and apparently blind, in which condition he remained all night and was thought to be dying. It was noticed that his arm and leg of the opposite side frequently twitched. The next morning, though still in stupor, he could be aroused. He was emaciated, tongue coated, yawned frequently, eyes wide open, temperature 99.3 degrees, pulse 62. There was slight lateral deviation of the eyes to the opposite side. Pupils were equal and responded to light. The pupil on the unaffected side was observed to dilate more widely than on the side of the lesion. There was marked right optic neuritis, with beginning left optic neuritis. Absence of both patellar reflexes.

**Operation.**—Half an ounce of pus was found in cerebellar tissue near the posterior surface of the petrous portion of the temporal bone. The finger was introduced. Cavity held open by the *forceps, when the latter accidentally slipped,*

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[37] Coates, G. M.: Temporo-sphenoidal Abscess, Double; *Annals of Otology, Rhinology and Laryngology,* June, 1917, Vol. 26, p. 408.

Two cases:

CASE I

Temporo-sphenoidal; no symptoms whatever; discovered by accident by bulging of dura; sterile pus; chronic.

CASE III

Two abscesses; no stalk; 1 cm. of cerebral tissue between two cavities; both apparently acute. History not explicit enough to say whether or not it was metastatic; could not have been caused by trauma as patient died on table.

*and the flow of pus stopped. Careful search failed to re-discover the cavity.* A drainage tube was introduced along the tract. During the next week the boy did well, was rational, no deviation of the eyes, co-ordination normal, but at times he would cry out, and there was involuntary defecation and urination. Optic neuritis increased in the left eye, and there was nystagmus when the eyes were moved toward the side of the lesion. Seven days after operation, the pulse and temperature being normal, paresis of the arm of the same side developed; the eyes deviated to the opposite side. On the tenth day the wound was explored with the finger, but nothing was found. On the fourteenth day after the operation he was in the same condition as primarily; pulse 60, respiration 14, complete paralysis of the arm and hand of the same side.

**Second Operation.**—On exposing the original sinus tract *a large abscess cavity was found,* running toward the median line; two ounces of pus were evacuated, after which *the hernia, which had developed during the first week, disappeared.* During the following three weeks the patient was so violent as to require restraint, and screamed frequently.

Discharged from hospital; the sinus was healed and remains so though on two occasions a small opening has developed over the site of the wound. His mental condition is very poor, though his mother says he is as bright as he ever was.

**Present Condition.**—Perfectly well; complains of headache. Served with American Expeditionary Forces in France.

## ACUTE BRAIN ABSCESS.

In acute intracerebral abscess the outstanding pathological process is acute cerebral tissue death, with intense œdema, and but slight protective limitation, the rapid formation of the abscess precluding the formation of a protective capsule. As the technic of the surgical treatment of chronic brain abscess with a capsule utilizes the capsule in the thorough evacuation, cleansing and obliteration of the abscess, the surgical technic of acute brain abscess must be largely modified to adapt itself to differences in the pathological processes.

Recovery in acute intracerebral abscess is much rarer than in brain abscess with a capsule, and yet I am persuaded that with a proper operative regard for the differences in processes, the mortality from acute abscess should not be much higher than in chronic cases.

**Pathology.**—The pathological changes in an acute brain abscess are the same as those occurring in an abscess formation in any part of the body, altered only by the special structure involved. Following the infection we have the same death of tissue and the same protective processes that occur elsewhere.

A microscopic specimen exhibits an inner zone of dead tissue and pus, necrosis from strangulation, and leucocytosis. If the infection is virulent enough to break down completely any attempted resistance of the part, there follows an extension of this process—

an extending death of tissue—and beyond, in the still living cerebral tissue, an intense œdema with thrombosis of the vessels, and hemorrhage. In such a case death of the patient occurs within a few hours of the original infection: fulminating infective encephalitis.

When the infection is not so virulent, protective processes are present. In the microbiotic zone, in which the bacteria and their toxins have caused a disappearance of the nerve cells, the glial cells, although altered, are still present, and external to this zone there is an area of round cell infiltration—from which the leucocytes have been called from the blood vessels to the brain for its protection—associated with vascular thrombosis and with a meshwork of fibrin.[38]

The microscopical picture of an acute brain abscess gives the general impression that the chief limiting process is thrombosis of the vessels, and hemorrhages, just as the formation of a new connective tissue is the chief limiting process in chronic brain abscess.

In this area of infiltration from the lymphocytes, the nerve cells are also dying or dead, but the neuroglia on the other hand show a certain amount of proliferation. Blood vessels now rapidly bud in this zone, each blood vessel simply being a blind endothelial tube from which fibroblasts spring.

**Surgical Pathology.**—If an acute abscess is now attacked by the surgeon, and during the evacuation and the subsequent introduction of a drain hemorrhage from the vessels occurs, the delicate protective mechanism is broken down, the œdema of the adjacent cerebral tissue is greatly increased, the inflammatory process acquires a fresh momentum from the trauma of the operation and the irritation of the drain, and death is apt to follow rapidly. (See Figs. 23, 24.)

**The Surgical Aims** should be limited to, (1) the location of the abscess; (2) the relief of compression by as complete evacuation of the abscess as possible *without trauma to the cerebral tissue;* and, where the suppuration is intracerebral, to (3) the establishment of an area of protective adhesions into the abscess cavity.

**Methods of Treatment.**—A clinical diagnosis of acute abscess without capsule having been made, one of two methods must be adopted. Either, (1) exploration through a large dural opening, followed by complete evacuation if a piarachnoid abscess is located, or, if an intracerebral abscess, by as complete evacuation as is possible through the searcher, with tight closure of the dura excepting such as is sufficient to admit of the exit of the searcher; or, (2) a

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[38] Hassin, G. B.: Histopathological Studies in Brain Abscess; *Medical Record*, January 19, 1918, Vol. 93, p. 91.

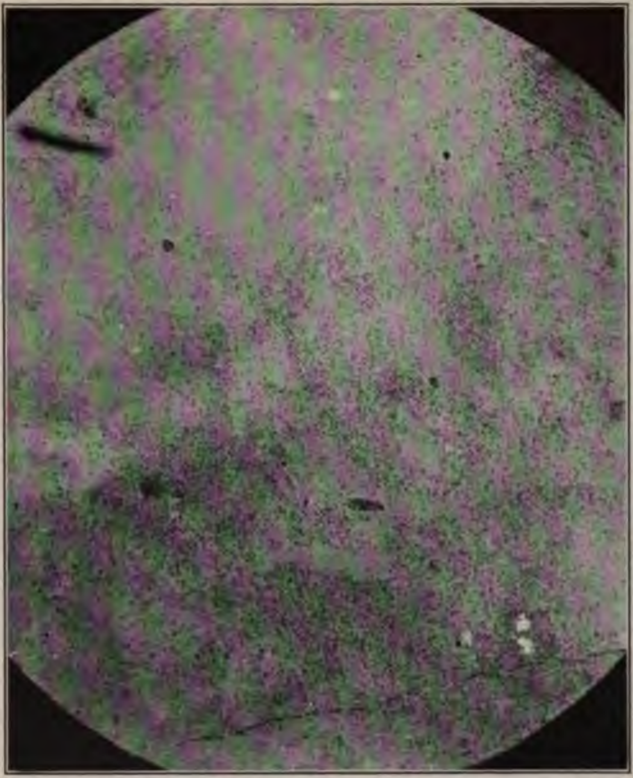

FIG. 23.—Microphotograph showing intense œdema, round cell infiltration and multiple hemorrhages in an acute cerebral abscess. Death 24 hours after evacuation, without return of consciousness.

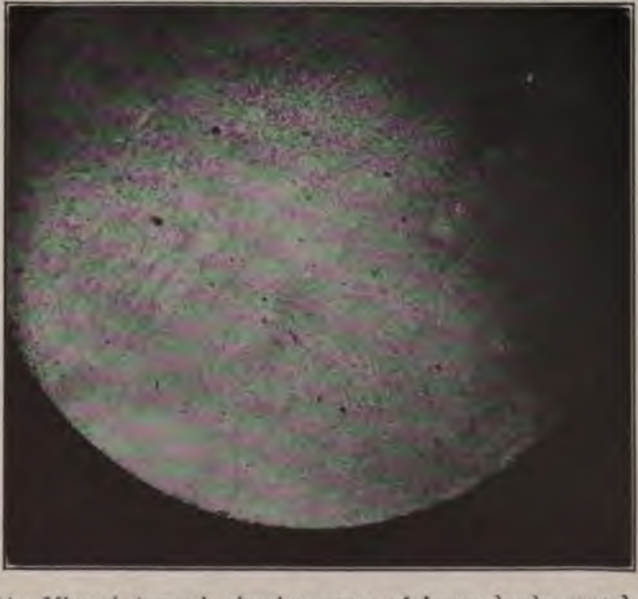

FIG. 24.—Microphotograph showing areas of hemorrhagic encephalitis, following evacuation of an acute brain abscess.

hollow needle puncture through the intact dura, the primary aim of the puncture being the location of the abscess and the establishment of a tract of protective adhesions through the piarachnoid into its cavity.

Were it possible uniformly to differentiate *acute non-capsulated intra-cerebral,* from *piarachnoid abscess* prior to operation, the puncture method would be preferable, since in acute intracerebral abscess the puncture, with but slight damage to the cerebral tissue, partially relieves the compression by the partial evacuation.

Unfortunately our diagnostic methods do not allow of such accurate differentiation, and if the puncture method is uniformly adopted many superficial piarachnoid abscesses will fail of localization, as has been the author's repeated experience. Consequently he has for years advocated and practised a dural opening large enough to admit a thorough inspection of the adjacent piarachnoid; and in case a piarachnoid abscess is not found, exploration into the brain substance without injury to a cortical vessel, with *partial* evacuation of the abscess and tight closure of the dura around the exploring instruments, which may or may not necessitate a second operation for complete evacuation, cleansing, and obliteration after the capsule has been found.

In 1914, before the New York Academy of Medicine, the author recorded:—"I am persuaded that the operation for acute intracerebral abscess, especially if situated more than two or three centimeters from the surface, should be conducted in two stages. My opinion is based upon almost a complete failure to effect a cure by means of exploration and introduction of drainage material into the brain in cases of acute, subcortical, intracerebral abscess.

Post-mortem examinations of acute intracerebral abscesses so treated revealed intense œdema and multiple punctate hemorrhages, while the drain was invariably found lying in the brain itself, entirely outside the abscess cavity, thus only exaggerating a previously bad condition. In my opinion it is better not to introduce drainage material at all than to damage brain tissue in an unsuccessful attempt to drain the cavity. When the operation is undertaken in two stages the first procedure should consist simply of the evacuation of the cavity with a minimum of traumatism.[39]

CASE XIII.

**Mrs. H.:—Acute Adjacent Temporo-sphenoidal Lobe Abscess.** Right middle ear disease, without discharge two weeks previously.

**Present History.**—Fourteen days previously suddenly had a severe pain in the right ear, accompanied by a chill. *No discharge.* Pain lasted twenty-four hours and then stopped. Temperature about 102 degrees. Few days later:—temperature ranged from 102 to 105 degrees; respiration, 32 to 36; pain in back of head recurred and persisted. Consolidation of lung was diagnosed, although no cough was present.

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[39] Lemaitre, F.: "L'Exclusion des espēces sous-arachnoidiens" (Exclusion of the Subarachnoid Spaces; application to the treatment of cerebral and cerebellar abscesses of otitic origin, and in a general way to brain surgery); *Revue de Chirurgie,* 1919, Tome LVII, p. 497.

A few days before examination there were mental symptoms; pulse slow; patient delirious at times, at other times more or less lethargic; temperature normal. No vomiting, but nauseated once—thought to be due to tobacco smoke in the room. No dizziness. Temperature irregular, ranging from normal to 103 degrees.

Examination on fourteenth day of illness:—patient more or less lethargic; can be aroused and answers questions well—it seems as if she answers too well, for although she keeps her eyes closed she is conscious of all that goes on around her. Right optic disc margin slightly indistinct, veins full; left margin clear, but veins full. Hears well with both ears.

Slight weakness of left arm, which appeared for first time at this examination. Has a very bad odor to her breath. The membrane of her mouth is swollen. She has no Kernig; no Babinski. Temperature which had ranged from normal to 101 degrees in last few days, suddenly rose to 103: slight chill. Lumbar puncture gave high cell count; no bacteria.

**Two Days Later.**—In deep coma; cannot be aroused; incomplete paralysis of left arm. During last two days several attacks of choking and cessation of respiration.

**Operation on Sixteenth Day of Illness.**—As there was some doubt whether patient was suffering from encephalitis lethargica or brain abscess, the only ear symptoms recorded having been pain which lasted twenty-four hours and had never been followed by discharge, it was decided to explore the mastoid first, and if evidence of disease was found, then to explore the brain. The mastoid antrum and a few cells were found to contain granulations and a few drops of pus. (Smear and culture from cells and antrum negative.)

Through a clean field above the mastoid and ear the bone under the temporal muscle and over the squamous portion of the temporal was removed and the dura exposed for an area about two inches in length by one and one-half inches in breadth. A small opening through the dura failed to reveal pus in or on the surface of the brain. A puncture of the brain through the opening, with hypodermic needle at depth of one inch from the cortex revealed thin pus. On account of small size of needle the flow was very slow and soon stopped altogether.

Needle left, and held in position by numerous small pledgets of wet cotton pyramided around it. Muscle and skin closed except for pyramided area.

At the beginning of the operation the blood pressure was 170, rising to 190 and falling immediately to 155 upon the partial evacuation of the abscess. Culture from pus from brain revealed streptococcus.

**Two Days after Operation.**—Patient has regained consciousness, recognizes objects and talks, but is still very lethargic. Left arm and leg both almost completely paralyzed. Slight papillœdema of both eyes. No drainage through the needle, but considerable over and around it, as if it had discharged for some time and then stopped. On passing wire through needle there was no pus. Patient gradually improved. Moved fingers of left hand and toes of left foot for first time in several days.

**Subsequent History.**—During the subsequent treatment of the abscess it was twice lost, and refound because of filling again with pus. A larger drainage tube was at last properly placed within the abscess cavity. The papillœdema persisted for over six weeks after the operation. The patient ultimately completely recovered.

**Comments.**—In this case we caused (1) slow evacuation, thus relieving the compression; then (2) the establishment of a small tract; later (3) the enlargement of the tract and again evacuating. During this treatment there was opportunity for the acute abscess to be converted into (4) a chronic abscess with a capsule. The continuation of the papillœdema may have been due to the lack of a wide decompression, or a displacement of the brain bulk may have been a factor.

The abscess was certainly touched at its side and not in the center—the collapse of its walls twice causing the loss of the cavity, which was not found again until it had refilled with pus, thus demonstrating the necessity of as complete evacuation as possible in acute as well as in chronic abscesses.

The leucocytosis three days after the operation was 24,000, with 83% polymorphonuclears. It rapidly fell to normal—6200 to 8800—in spite of the abscess refilling twice, showing that routine cell counts are of very limited value after evacuation as to the progress of the case.

The object of the operation is the evacuation of the abscess and the collapse of its walls, with the least possible associated trauma to the cerebral tissue. By the evacuation some of the infective material is removed and this, with the associated relief of tension, may allow time for the tissue to develop resistance and the formation of a capsule. Later, should the symptoms recur, the area of suppuration being now limited, the abscess may again be explored, when, as in one of my cases, the meninges will be found walled off—a very important aid. The abscess may now be fully evacuated and drainage material introduced.

**Technic.**—In exploring for an acute abscess, three inches of the end of a soft rubber catheter containing two holes, with a flexible metal stylet, should be used. The stylet should occupy but a small part of the lumen of the catheter, so as to allow the thin pus to flow through the catheter with the stylet in place. On locating the abscess the greatest care must be taken not to displace the catheter. All pus presenting is collected by a suction apparatus. After pus has ceased flowing the stylet is removed and the catheter cut off flush with the skin, to which it is attached by a silk suture. The patient should then be inverted. If the abscess is very acute the catheter may be withdrawn; if of longer duration, the dural opening should be entirely closed except for the small opening through which the catheter passes and which it should entirely fill. The skin opening should be tightly closed, except for a similar opening.

The technic of the second complete evacuation is that adopted in chronic intracerebral abscess.

### CLOSURE.

In all cases of brain abscess, chronic or acute, the abscess having been entirely evacuated, the brain should be as completely covered as possible. If the technic of evacuation has been perfect, the dura can be almost closed, the small area which carries drainage into the abscess cavity alone being unprotected by the dura.

Many otologists and surgeons disregard the fact that all drainage material within the dura is dangerous, especially if it is absorbent. To place dry gauze over an exposed area of piarachnoid over which a dressing is introduced, is simply to invite disaster. As a proof of this, witness the very extensive superficial brain necrosis often found at autopsy after so-called "free" drainage.

If a drainage tube or strip of rubber tissue has been introduced into the abscess cavity, a tract for the drainage material should be made to the nearest external surface, through dura, bone, and skin. Over all the remaining area, dura, bone and skin should be covered, unless the soft parts are so wet that it is thought advisable to place a drain between the dura and the bone.

It is not an uncommon experience to find, at the end of an intradural manipulation, that the protruding brain prevents the complete closure of the dura. When it does occur, the piarachnoid should be covered with a piece of fascia lata stitched firmly to the dural defect, over which as much as is possible of the bone flap should be placed, and the fascia, muscle and skin lightly closed.

It is best to leave all drainage undisturbed for several days, during which an organized wall is being built along the tract, thus placing the tract in reality outside the brain tissue. Further manipulations along this tract, such as the re-introduction of drainage or of the encephaloscope, should be conducted, if at all, without the slightest injury to this wall. During recent years, in my successful cases, the drainage material has been left entirely alone for rather long periods. As it is slowly expelled by the brain, it is carefully cut off, care being taken to anchor the end. After a period of from two to five weeks, depending upon the depth of the abscess, its size, duration and amount of discharge, the last piece of drainage is removed.

### *Additional References to Double Abscess.*

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Urbantschitsch, E.: Zweifacher rechtseitiger Schläfenlappenabszess: Operation: Heilung; (Double Right-sided Temporal Lobe Abscess; Operation; Recovery); *Wiener Klinische Wochenschrift*, 1916, Bd. XXIX, No. 47, S. 1507.

### *Additional References to Temporo-Sphenoidal Brain Abscess.*

Ruttin, E.: Schläfenlappenabszess mit Infektion des rechter Unterhorns, Pyocephalus, Meningitis, Exitus; *International Centralblatt für Ohrenheilkunde*, 1909-10, Bd. VIII, S. 379.

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Bodkin, H.: A Case of Cerebral Abscess of Otitic Origin; *British Medical Journal*, 1917, I, p. 510.

Nauwerck: Zur Kenntniss des chronischen, traumatischen Hirnabszesses; *Münchener medizinischer Wochenschrift*, 1917, Bd. LXIV, S. 109.

# CHAPTER V.

# METASTATIC ABSCESS.

## CLASSIFICATION.

The accepted classification of brain abscess of otitic, rhinitic, or metastatic origin calls for modification. Many abscesses originating from the ear and the nasal sinuses are pathologically and clinically metastatic and should be so classified, their symptomology permitting differentiation from the usual adjacent (secondary) abscess of aural or nasal origin.

The classification of brain abscess into Adjacent (secondary to aural or nasal suppuration), and Metastatic (from lungs, heart, ear, etc.), as suggested elsewhere in this monograph (Chapter III., p. 17), is based upon two distinct types of pathological process:—the *adjacent* type, invading the brain by direct extension either through tissue suppuration, or by way of a thrombophlebitis, or perivasculitis, and the *metastatic* form, which is of hematogenous origin.

From a pathological standpoint a brain abscess which originates from an infection *free in the blood stream* is truly metastatic whether the primary focus be situated in the ear or in the lung. The classification into Adjacent, Metastatic and Traumatic will explain many reported cases otherwise enigmatic—such as abscess of otitic origin of the opposite hemisphere—while clinically it will prove of assistance in diagnosis and surgical treatment.

## DEVELOPMENT.

**Adjacent Abscess.**—Adjacent brain abscess, as the name implies, is anatomically adjacent to the primary focus of infection, viz., mastoid antrum and middle ear, labyrinth, or nose, to which it is directly connected by a pathological process. Its position within the brain, therefore, must be more or less definite, being prescribed by anatomical relationship. The infection from the ear or nasal sinus extends into the cerebral tissue by continuity of tissue suppuration, or necrosis, or by contiguous extension through a venous wall or a perivascular space—retrograde thrombophlebitis or perivasculitis.

Retrograde thrombophlebitis of a cerebral or pial vein follows the course of thrombotic formation in general; it advances in the wall of the vein—a phlebitis—or in a perivascular space. The phlebitis or perivasculitis is followed first by a parietal, and later by an obliterating thrombus. A blood-stream infection, if present at all, is secondary to the thrombus formation. In accordance with thrombotic formation in general, the process is comparatively slow.

The chief surgical and diagnostic difference between the formation of an abscess from retrograde thrombosis and from a metastasis is based on the fact that in the former the protective mechanism of the brain has time to assert itself while the circulation is still active and before the death of cerebral tissue takes place. If the symptoms are present at all at this time, they must of necessity be trivial since they are due to a normal reactive process. Likewise, the clinical symptoms of adjacent abscess develop gradually; they are never apoplectiform.

**Metastatic Abscess.**—Metastatic abscess follows the occlusion of a cerebral vessel by an infected nidus circulating freely within the blood stream itself. Bacterial infection of the blood stream alone apparently is not sufficient to produce a brain abscess. In the numerous experiments of Weed and his associates [1] with direct blood-stream infection, only one superficial abscess (from infection of a perivascular space) developed. The blood stream is intolerant of the presence of micro-organisms within itself. Following injections of micro-organisms into the blood stream, the blood soon eliminates them and rapidly becomes sterile again. An additional factor, apparently, is necessary; this is probably supplied by a minute septic nidus small enough to pass through the capillaries. Toxæmia causes great damage to the intima of the blood vessels because of the slowness of the venous circulation, and a toxæmic nidus finds a favorable condition in the intima for the rapid growth of a thrombus which soon may become of embolic nature.

**Metastatic Vascular Occlusion.**—The onset of metastatic brain abscess is sudden, apoplectiform, because the bacteria, suddenly closing the vessel, cause apoplectiform symptoms—a sudden, violent headache, transient dizziness, projectile vomiting. These symptoms may be slight, and apparently transient, but the sudden vascular occlusion causes immediate nutritional death of the cerebral tissue.[2]

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[1] Weed, L. H., Wegeforth, P., Felton, J. B., and L. D.: Meningitis Produced by Intravenous Inoculation; *Monographs Rockefeller Institute for Medical Research*, March 25, 1920, No. 12, pp. 57-112.

The focus of original infection is generally distant from the brain—in the lungs, the heart, or the pleura—but numerous cases are on record of the infecting nidus originating from blood vessels contiguous to the ear. In a case reported by Berens,[3] infection originated from thrombus of the internal carotid artery; in one of the writer's from the lateral sinus. Cases resulting from abscess at the surface of the body are not of common occurrence, but Kutzinski and Marx [4] report one in which abscess of the frontal lobe apparently resulted from a purulent process in the finger. All such cases should be classified as metastatic. The usual explanation that metastatic abscess is always of embolic arterial origin has not been substantiated by the author in an examination of the post-mortem records.[5, 6]

Non-suppurative arterial occlusion is comparatively frequent, but it does not occasion abscess formation although the symptoms may simulate those of abscess when associated with a suppurative process.

CASE XIV.

A. S.: Thrombosis of Cerebral Vessels. Child, aged seven months.

**History.**—The father died from tuberculosis when the child was only a few months old. The child seemed perfectly well until one day it suddenly cried, had a convulsion, and was brought to hospital with twitching of its right upper lid, corner of mouth, and right arm and leg, with a lateral deviation of its eyes to the left. O. E. negative. On following day spastic paralysis of right arm and leg. Developed otitis media in left ear; right ear began discharging about four days after admission to hospital, with swollen area over mastoid. Nystagmus to left, continued lateral deviation of eyes to left. Continued twitching of right arm and leg; facial paralysis; frequent slight convulsions, followed by profuse perspiration. Increasing coma. Temperature normal until three or four days before death, when it ran to 105 and 108 degrees. Child died three weeks after admission to hospital.

**Diagnosis.**—Inherited tuberculosis from father, followed by acute tuberculosis, with thrombosis of cerebral vessels from tuberculous infection.

**Autopsy Findings.**—On opening skull the left frontal lobe was found softened—not an abscess, but nutritional disturbance from vascular occlusion. The softening extended over to right frontal lobe, with portions of infiltration where blood had been effused into the brain. The right hemisphere showed signs of increased intracranial pressure. Embolus in anterior cerebral artery, causing degeneration of the whole frontal lobe. Around the place where the thrombus occurred there was hemorrhagic infiltration. Examination of right ear showed grumous material in the middle ear and antrum; area of bone around seemed softened. The left ear also contained grumous material.

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[2] Moore, John T.: Blastomycosis. With Report of Case Dying from Abscess of the Brain; *Sur., Gyn. and Obs.*, December, 1920, pp. 590-594.

[3] Berens, T. Passmore: *Annals of Otology, Rhinology and Laryngology*, 1913, Vol. XXII, p. 448.

[4] Kutzinski, A., and Gerichtsarzt, Marx: Hirnabszess als Folge peripherer Körpereiterung nach einem Unfall; *Monatschrift für Psychiatrie und Neurologie*, October, 1914, S. 255.

[5] Schorstein: Lancet, 1909, II, p. 843.

[6] Cayley: Abscess of the Brain Associated with Bronchiectasis; *Transactions of the Pathological Society of London*, 1884, Vol. XXXV, p. 12.

**Metastatic Abscess of Venous Origin.**—The vast majority of, if not all, metastatic brain abscesses are undoubtedly of venous origin, for here, as in embolus of thrombotic origin in general, conditions are favorable for its development, while the rapidity of the arterial flow, assisted by the brain's protective mechanism, tends to prevent the lodgment and growth of a small infected nidus in the arterial circulation. This view explains and is substantiated by the relative infrequency of brain abscess in ulcerative endocarditis, in which large numbers of infected nidi are carried to the brain by the arterial circulation.

**Origin of Thrombotic Nidus.**—Parietal venous vegetations are more frequently associated with mastoiditis and sinus phlebitis than is generally supposed, the vegetations grossly resembling those of the heart in acute endocarditis.

CASE XV, R.:

Meningitis from Labyrinthitis; with Beginning Thrombi and Vegetating Thrombus in Longitudinal Sinus; Male.

**History.**—Always subject to headache. Had had an attack of influenza, followed by sharp pain in right ear. Spontaneous rupture of drum membrane. Ear continued to discharge for about six weeks; during entire time patient swayed to right and complained of pain on right side of head. Lost considerable flesh. Attended to his business until physician ordered him to country. While away, had a distinct chill and severe headache, and vomited during the night. Lumbar puncture gave an excessive amount of fluid under pressure, slightly turbid or yellowish, looking like urine and containing a large number of polymorphonuclear leucocytes; apparently sterile. Totally deaf in right ear. Examination revealed no discharge from this ear. A teat-like projection was seen in lower quadrant. Marked nystagmus at outer corner. O. E. gave fulness of the veins; no spontaneous pointing deviations.

**Operation.**—Mastoidectomy. The whole mastoid was sclerosed, being absolutely solid. Exploration of the cerebellum negative.

Patient was in deep coma the day following operation, and died three days later with marked symptoms of suppurative meningitis.

**Autopsy Findings.**—The bone was hardly cancellous to the naked eye and was sawed with great difficulty. The piarachnoid was infiltrated with pus. The Pacchionian bodies were prominent alongside the longitudinal sinus. On opening the superior longitudinal sinus the cavity was found to contain a cluster of vegetations posteriorly to a plane corresponding to the occiput. These vegetations were not unlike those seen in the valves in bacterial endocarditis. Some were friable and could be torn off easily, the majority were fibrous, were well organized and could not be stripped off. They almost obliterated the lumen of the sinus. The right labyrinth, converted into one large, mouse-eaten cavity, contained a thick film of foul-smelling pus.

Smears made from the right middle ear, labyrinth, meninges, and some of the vegetations in the longitudinal sinus, showed streptococci. Sections of the longitudinal sinus where the vegetations occurred showed the wall to be thickened by fibrosis. The vegetations sprang from the intima and consisted of a stem of connective tissue in which there was a marked infiltration of small and large mononuclears and polyblasts. In some of the more recent ones polynuclears were abundant.

**Comment.**—Undoubtedly the severe pain in the back was due to lodgment of embolic portions of the sinus in the small vessels of the cord. Without having obliterated the sinus, the vegetations were being given off into the circulation all the time.

A venous sinus vegetation, if detached, need not necessarily go through the entire circulation before again entering the cerebral venous circulation. Arnold[7] has demonstrated experimentally that, on injection of bran into the large veins of the neck in case of interference with the normal intrathoracic negative pressure, particles of the bran are found in the opposite venous sinus. According to his experiments the venous circulation from the head toward the heart is reversed when a positive intrathoracic pressure is encountered. Mechanically, then, disturbance of the return venous circulation, such as an occluding thrombus or temporary compression from a tampon, may cause a reversal of the current from one venous sinus to that of the other side, there being no obstructing valves.[8] These factors, associated with a disturbance of the protective mechanism of the brain, render possible the lodgment and growth of the embolic thrombus. The protective mechanism of the brain is easily interfered with by circulatory disturbance, as demonstrated by Weed,[9] by the production of meningitis from a blood-stream infection by jugular decompression. Arnold's[10] observation provides an explanation of abscess of the brain on the opposite side from the ear lesion, such as that referred to by Japha,[11] Quimby,[12] Lossen,[13] and Maier,[14] or of any of the unusual sites of abscess formation in the frontal or occipital lobe from ear suppuration,[15] or in the parietal from nasal infection.

## FREQUENCY.

Abscesses of the brain of metastatic origin, while of relatively rare occurrence, are much more frequent than is generally recognized.

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[7] Arnold, J.: Ueber Rückläufigen Transport; *Archiv. für Pathol., Anat., und Physiol.*, 1891, Bd. 124, S. 385.

[8] Knott, J. F.: The Cerebral Sinuses and their Variations; *Trans. Int. Med. Congress, London*, 1881, Vol. I, p. 191.

[9] Weed, L. H.: Production of Meningitis by Release of Cerebro-spinal Fluid; *Jour. Am. Med. Assoc.*, 1919, Vol. 72, p. 191.

[10] Arnold, J.: *Loc. cit.*

[11] Japha, A.: Zur Diagnostik der Herderkrankungen des Gehirns; *Zeit. für Prak. Aerzte*, 1898, VII, 701.

[12] Quimby, Wm. O. G.: *Personal Communication.*

[13] Lossen, W.: Beiträge zur Diagnose und Therapie der Kleinhirnabscesse (Case I); *Beiträge z. Klin. Chirurgie*, 1903, Bd. 39, S. 804.

[14] Maier, M.: Erfahrungen über den otitischen Hirnabscess; *Archiv für Ohrenheilkunde*, 1914, Bd. 95, S. 163.

[15] Berens, T. Passmore: Abscess of Frontal Lobe of the Brain, with Exhibition of Specimens; *Trans. Amer. Otol. Soc.*, 1913, Vol. XIII, Part I, p. 68.

About twelve per cent. of these abscesses are caused by suppurative pulmonary disease;[16] approximately an equal percentage have their origin in pyemic foci other than those situated in the lungs; and a small number are due to amœbic dysenteric disease of the liver;[17, 18] while in a certain proportion of cases the cause is unknown.[19] A number of so-called "idiopathic" brain abscesses have been reported,[20, 21] in which a careful post-mortem examination failed to disclose a primary cause in any other organ (see page 110), although the demonstration of the fusiform bacillus in the tonsils[22] and of the fusiform bacillus and anaërobic streptococci in the lungs has recently diminished the number.

In 1915 the author reported a metastatic abscess originating in the ear; since that time an analysis of recorded cases of brain abscess has confirmed the opinion that certain abscesses originating from the ear and nose are of a clinical type different from the usual adjacent abscess. In common with all metastatic abscesses of the lungs, the liver or the heart they have an apoplectiform onset, and, not being limited by the anatomical relationships that more or less restrict an adjacent abscess, they may be situated in any part of the brain.

Examination of cases of abscess of the frontal lobe of aural origin, of the temporo-sphenoidal lobe of the opposite side of the cerebellum of nasal origin—in fact of *all the cases* of abscess not adjacent to the suppuration—substantiates this view.[23, 24, 25] Groth[26] collected thirty-three cases of abscess of the brain due to pulmonary affections, and in his review he endeavors to explain the frequency of brain abscess in suppurative pulmonary disease on the one hand, and its relative infrequency in suppurative endocarditis on the other, by attributing it to mechanical conditions in the circulation. This, however, may be more satisfactorily explained by assuming a thrombotic, not an embolic origin.

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[16] Meyer, R.: Ein Fall von gangranöser Lungentzündung mit metastischen Abszessen in Leber und Hirn und Bemerkungen zur Symptomatologie und Diagnose des Hirnabszesses; *Berlin Klin. Wochenschrift,* 1868, Bd. 5, S. 429.

[17] Couteaud: Abscès metastiques de l'encéphale en rapport avec les suppurations hépato-pulmonaires; *Revue de Chirurgie,* July, 1913, Tome XXXIII, No. 7, p. 91.

[18] Armitage, F. L.: Amœbic Abscess of Brain; *Journal Trop. Med. and Hyg.,* April 15, 1919, p. 69. (States that 43 out of 45 cases followed liver abscess.)

[19] Gowers, W. R.: Diseases of the Nervous System; London, 1893, Vol. II, p. 470. (States fifteen per cent.)

[20] Henderson, J.: A Case of "Idiopathic" Cerebral Abscess; *Lancet,* London, May 31, 1913, I, p. 1525.

[21] Reynolds, Cecil E.: A Case of Brain Abscess; *Jour. Amer. Med. Assoc.,* February 7, 1914, Vol. 62, p. 449.

[22] Dick and Emge: Brain Abscess Caused by Fusiform Bacilli; *Jour. Amer. Med. Assoc.,* February 7, 1914, Vol. 62, No. 6, p. 446; *Trans. Chicago Path. Soc.,* 1914, IX, p. 995.

[23] Tylor, Christopher: The Paths of Encephalic Infection in Otitis; *Brain,* London, 1912-13, Vol. XXXV, p. 109.

[24] Berens, T. Passmore: Brain Abscess with Unusual Features; *Annals Otol., Rhinol., Laryngol.,* 1910, Vol. 19, p. 218.

[25] Lombard, Bloch A., Moulouguet, A.: Un cas d'abscès du lobe frontal du côté opposé à une otite suppurée chronique; *Annal des Maladies de l'Oreille;* 1914, Tome-90, p. 749.

[26] Groth, W.: Beitrag zu den Metastatischen Hirnabszessen pulmonalen Ursprungs; *Inaugural Dissertation,* Berlin, 1910.

It is a clinical fact of great importance that while suppurative diseases of the lungs, particularly bronchiectasis,[27, 28] frequently cause abscess of the brain, suppurative endocarditis rarely does so, although the size and directness of the arteries from the heart to the brain would lead one to expect abscess of the brain to be a frequent complication of suppurative endocarditis. Such, however, is not the case;[29] while metastatic abscess of the kidneys and spleen are common in suppurative endocarditis, brain abscesses only infrequently occur, but one case having come under the author's observation. According to Sperling,[30] in seventy-six cases of endocarditic emboli from the left heart, metastatic abscess occurred in the kidneys in fifty-seven, in the spleen in thirty-nine, and in only fifteen in the brain.

Abscess of the brain secondary to suppuration in the lungs would appear to be primarily thrombotic or venous in origin, the brain being peculiarly susceptible to such infections; whereas abscess resulting from suppurative endocarditis may be primarily embolic, against which the brain may possess a resistance not present in the spleen and kidneys. Roulland's successful case[31] demonstrates that prompt surgical procedure offers a fair prospect for recovery in metastatic abscess of pleuro-pulmonary origin.

**Location.**—In pulmonary disease the left side of the brain is the site of metastatic abscess three times more frequently than is the right (Gowers), and the frontal lobe is especially apt to be the site of the abscess.[32]

In proposing exploration in metastatic brain abscess the surgeon is confronted with a question of the greatest importance in relation to the future surgery of this condition:—"In the event of an abscess being found, will the paralysis be cured?"

If the pathology of metastatic brain abscess is that of an infective embolus carried from a distance and deposited in a terminal cerebral artery, acting simply as a local embolus with a secondary infection of the surrounding tissue, and if the suppurative embolus involves the

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[27] Schorstein: Cerebral Abscess in Diseases of Lung: Lancet, London, September 15, [illegible], p. 843.

[28] [illegible] P. et [illegible]: Abcès du cerveau [illegible] par un traumatisme chez un sujet porteur de dilatations bronchiques: Bull. et Mém. Soc. Méd. des Hôp., 1920, III. Series No. 27, p. 1166.

[29] Krause: Surgery of the Brain: [illegible], Vol. III, p. 924.

[30] Sperling: Inaugural Dissertation: 1872.

[31] Roulland, H.: Abcès métastatique du cerveau au cours d'une suppuration pleurale. Trépanation, drainage de l'abcès. Guérison. Paris Chirurgical, 1917, Tome IX, p. 613.

[32] [illegible]: Trois cas d'abcès du cerveau consécutifs à des abcès du foie; [illegible] de Médecine, [illegible], Tome [illegible], p. [illegible].

same vessel from which hemorrhage generally occurs,—the so-called "artery of cerebral hemorrhage"—then the draining of the abscess will *not* relieve the paralysis, as these branches, being terminal, have no anastomotic communication in non-suppurating vascular disease.

In answering this question it should be realized that the paralysis or loss of function caused by abscess of the brain from direct extension from adjacent suppurative lesions invariably disappears after evacuation; and if metastatic abscesses of the brain are thrombotic and deposited in the veins and not in the arteries, then drainage should have a decided effect on the relief of the paralysis.

Cadwalader[33] has shown that in apoplexy the spontaneous intracerebral hemorrhages are apt to be large, and that when this is so they almost invariably cause death; while complete recovery from cerebral vascular occlusions and later softenings frequently takes place.

**Multiple Metastatic Abscesses.**—Metastatic abscess may be multiple, due to the involvement of the blood stream itself. In this respect it differs from adjacent abscess which, from its etiological pathology is single, at least originally, unless complicated by another process, such as trauma. On account of the tendency of suppuration to extend along the white fiber tracts, leaving the better nourished gray matter, the abscess may have prolongations which have frequently been mistaken for separate abscesses. It has been frequently observed, however, that metastatic cerebral abscesses secondary to suppurative disease of other parts of the body are usually multiple. This has been the author's experience.

CASE XVI, T. V.:

Autopsy 1280. *Specimen:* Section of brain, showing small abscess beneath cortex; very little attempt at encapsulation. A similar small abscess was found in cortex, extending to piarachnoid and producing a suppurative meningitis.

*Cause of Death:* Multiple abscess of brain, due to pneumococcus bronchiectasis.

The author has seen as many as five brain abscesses at postmortems, and separate abscesses to the number of thirty have been reported. Belief in the multiplicity of such abscesses has deterred surgeons from operating and consequently the number of operations undertaken for their relief has been very limited. Examination of the literature of the last twenty-five years shows, however, that forty-five per cent. of all metastatic brain abscesses were solitary.[34] In view of this it is justifiable for the surgeon to explore the brain in all cases of general pyemia presenting localizing apoplectiform cerebral symptoms, especially if the original focus of infection can be drained, as when the abscess is secondary to empyema, psoas abscess,[35] or abscess of the liver, phlebitis of the lateral sinus, or similar conditions.

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[33] Cadwalader, William B.: A Comparison of the Onset and Character of the Apoplexy Caused by Cerebral Hemorrhage and by Vascular Occlusion; *Jour. A. M. A.*, May 2, 1915, Vol. LXII, p. 1385.

TREATMENT. *Surgically,* then, metastatic abscess should be divided into two classes: (a) abscesses in which the original infection is removable—all such abscesses should be operated upon;—and (b) abscesses in which the primary cause is strictly hematogenous, and which are beyond surgical intervention.

### LIMITATION OF METASTATIC ABSCESS BY CAPSULE.

It is a frequent statement that metastatic abscesses are characterized by absence of capsule. A superficial examination of the reported cases would apparently substantiate this, as a large number are without limiting membrane; but a careful analysis will demonstrate that the absence of a capsule is in no way inherent in the pathological process itself. The capsule is absent because in a large proportion of cases there is not sufficient time for its development before the death of the patient. Especially is this the case in metastatic abscess of pulmonary origin, in which death generally follows within a few days the appearance of cerebral symptoms.[36]

Even in cases of slightly longer duration the suddenness of the cerebral involvement would necessarily allow insufficient time for the formation of a limiting membrane, whereas in an adjacent abscess of equal duration the forces for protection and limitation are marshalled long before the actual involvement of the cerebral tissue.

### FORMATION OF THE CAPSULE.

The thickness of the capsule will depend upon the age of the abscess, and—what is of equal importance—upon the virulence of the micro-organism and the tissue reaction necessary to limit the advance of the suppuration. In certain cases the extension of the suppuration apparently is easily limited. Cases have been reported in which, although the abscess was of long duration, the capsule consisted simply of a thin membrane. In a case referred to by Kölpin,[37] an abscess of three months' duration was bounded by *unchanged tissue,* and had not a capsule in the true sense. In this case, however, the man in the beginning had symptoms of cerebral involvement which persisted for a few days and then disappeared. The abscess, therefore, was probably a metastatic abscess of only seven days' duration and could not have had a capsule.

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[34] Bibrowicz, W.: Beiträge zur Klinik und Chirurgie der Hirnabszesse; *Beiträge zur Klinik und Chirurgie*, 1905, Bd. 47, S. 407.

[35] Saelhof, Clarence C.: Multiple Brain Abscesses Secondary to Bronchiectasis and Kyphoscoliosis; *Jour. Nerv. and Ment. Diseases*, April, 1920, Vol. 51, No. 4, p. 330.

[36] In Schorstein's nineteen cases death occurred in from three to twenty-eight days—an average of ten days. All of Schorstein's cases over seventeen days had a capsule. Frankel, Krause, and Saelhof's cases—twenty-four days—all had capsules.

All the symptoms of cerebral involvement must be taken into account in a consideration of the existence of a capsule. In Kölpin's case the abscess, even if it were of three months' duration, might or might not have had a firm capsule. However that may be, the protective tendency of the brain is toward the formation of a capsule, and in brain abscess continuity of cerebral symptoms, or the return of cerebral symptoms after a period of quiescence, generally means an encapsulated abscess.

A great deal has been written about the encapsulation of brain abscess. Körner [38] gives it as his opinion that the presence or absence of a capsule has no significance with regard to the age of an abscess, but a study of the cases in the literature would confirm the author's experience that the formation of a microscopically demonstrable capsule begins at about the seventeenth day of the cerebral involvement and in a rough way progresses with the age of the abscess.[39]

Cassirer [40] cites cases in which the capsule was found in from two to five days, although he admits that these were exceptional. His conclusion is that while the exact time of the formation of the capsule probably never will be settled definitely, a localized cerebral suppuration always means encapsulation.

Urbantschitsch [41] reports a case of temporo-sphenoidal lobe abscess which, probably after rupture into the first horn, was followed by an encapsulated empyema of the posterior horn.

CASE XVII, M. D.:

**Metastatic Brain Abscess of Otitic Origin, the Result of an Infective Vascular Occlusion, with Involvement of the Internal Capsule, Clinically Simulating Cerebral Apoplexy and Presenting Unusual Psychic (Toxic Delirium) Phenomena. Operation. Recovery.**

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[37] Kölpin: Zur Symptomatologie und pathologischen Anatomie des Hirnabszesses; *Deutsche Zeitschrift für Nervenheilkunde,* 1903-04, Bd. XXV, S. 465.

[38] Körner: Die otitischen Erkrankungen des Hirns, der Hirnhäute und der Blutleiter.

[39] See further discussion of formation of capsule, Part II, Surgical Pathology and Operative Technic, p. 47.

[40] Cassirer, Richard: *Archiv für Psychiatrie,* 1902-03, Bd. 36, S. 191.

[41] Urbantschitsch, E.: Schläfenlappenabszess mit abszessaktem Empyem des Hinterhons des linken Seitenventrikels; *International Centralblatt für Ohrenheilkunde,* 1909-10, Bd. 8, S. 554.

The salient features of this case may be summarized as follows: (1) Right-sided acute otitis media; (2) Mastoid operation, perisinus abscess with necrosis of the sinus wall and accidental opening of the sinus; followed by (3) Severe dizziness—probably serous labyrinthitis—and within thirty-six hours by an apparent "apoplectic stroke," with complete motor and sensory paralysis of the left side; (4) Erysipelas; (5) Gradual improvement and two weeks later a sudden increase in the symptoms; (6) Toxic delirium.

**History.**—Female, 53 years of age, unmarried. Previous good health. January 15, 1914, pain in right ear and dizziness; January 16, spontaneous rupture; January 18, paracentesis; chill. January 19, severe chill, temperature 101 to 105 degrees. January 20, two severe chills, followed by a temperature of 101 to 103 degrees.

**Diagnosis.**—Probable sinus thrombosis.

**Operation.**—On January 20 (five days after first pain in ear). Operative findings: Acute mastoiditis, with perisinus abscess; necrosis of sinus wall; free hemorrhage.

**Subsequent History.**—On the following day, January 21, great restlessness; temperature of 100 to 101 degrees, accompanied by dizziness and vomiting. During the night, when the patient attempted to raise a bowl of soup to her face with the right hand, she complained of a sharp pain in left (opposite) wrist and knee. According to the nurse she was able at that time to move the hand. A few hours later she was discovered to be completely paralyzed in the left arm and leg, with complete loss of sensation on that side.

January 23, temperature 101½ to 102 degrees. Fifty-four hours after operation, erysipelas extending on to face from region of affected ear. January 24, temperature 97.3 to 99.4 degrees. Erysipelas spread rapidly to opposite side of face and then subsided. January 25 to 29, temperature 98.3 to 100 degrees. Delirious at times. Violent pain in head, especially at night, necessitating the administration of morphine. Pain in right eye. Throbbing in left ear. Slight improvement in paralysis of arm and leg, enabling patient to raise arm to head. Slight delirium at times; irregular vomiting.

January 30 to February 3, temperature between normal and 101 degrees. Paralysis of arm considerably improved; arm raised to head with difficulty. Still severe pain in head and in right eye. *February 4, fifteen days* after operation, the temperature, which had been normal for five days, fell to 97.4 immediately rising to 101 degrees. Vomiting; sudden *increase of paralysis*, so that the arm and leg could not be moved; mental condition much worse. At this time the nurse noticed a loss of sensation in the left side of the face, the patient not feeling a basin when pressed against it, as she had previously done. Involuntary movement. Difficulty in swallowing. O. E. negative.

On February 6, seventeen days after operation, the patient was examined with the following findings: Complete motor paralysis of the left arm and leg. Paresis of upper part of right side of face (which may have dated from the time of operation, but which had been obscured by the erysipelas). Complete sensory paralysis of the left side (of face, left arm and leg). Left abdominal reflex absent. Left-sided hemianopsia. O. E. negative. Apparently a lateral deviation of the eyes toward the right side, although the patient moved eyes to left. Slight nystagmus on looking toward left. Right ear totally deaf; complained of "noise" in that ear and in the head, resembling "raining," "pounding," or "sea sounds." At times there were sounds in the left ear like the puffing of a locomotive. The right vestibular apparatus non-reactive to cold.

Patient drowsy, but could be easily aroused, when she recognized objects which, however, she was slow in naming. Although she *heard* with the left ear, she apparently did not hear promptly, as, when asked a question, she did not answer for a few seconds. (It is difficult accurately to describe this "delayed hearing": it suggests, but differs from the "slow cerebration" so frequently associated with brain abscess. It impressed one as a physical defect, such as ordinary deafness; while "slow cerebration" is obviously a mental defect.)

During the examination the patient was apparently perfectly normal mentally, but on being left to herself she gradually wandered into semi-delirium. She was garrulous and disorientated as to time and place. She tried to leave the bed with the desire to return home, from which she repeatedly asserted she had been removed for "white slave" purposes and taken to different places where proposals of a sexual nature had been made to her.[42] When told that she was at home she agreed, but insisted that she had been away. The paralysis of the arm also formed the basis for another fixed delusion. She asserted that she had lost her arm, or that it had been placed in a bureau drawer, and she frequently asked the nurse to bring it to her.

Blood pressure, 115 mm. Heart negative. Urine contained a slight amount of albumen; no sugar; specific gravity 1018.

February 8, Examination: Negative blood culture. Spinal fluid pressure slightly increased. Transparency, clear, watery. No sediment after centrifugalization. Globulin; moderate increase. Twenty-one cells, mostly polynuclears. Fehling's reduced. Bacteriological examination, sterile. Conclusion: Secondary protective meningitis, probably from brain abscess.

**Remarks.**—The cause of the paralysis, judging from its sudden onset, was apparently an apoplexy from hemorrhage or embolism, totally independent of the previous infective process. This view was further strengthened by the age of the patient—fifty-three years—and also by the fact that, so far as the writer is aware, there is no case in the literature of *sudden* involvement of the internal capsule of otitic origin. The more he thought of the case, the more apparent became the fact that the patient had had a virulent infection, which had rapidly caused involvement of the sinus wall, upon which had been superimposed an erysipelas with, probably, complete destruction of the inner ear. While the sudden onset of the paralysis pointed clinically to a vascular lesion, it was conceivable that such a virulent infection *might* cause a metastatic septic occlusion of one of the vessels to the internal capsule. The *exact* point of involvement in the posterior limb of the internal capsule was easily determined by the hemiplegia and hemianesthesia, associated with hemianopsia, and what was apparently *hemianacusia.*

An exploration was undertaken, largely because the sexual delusions were apparently becoming more firmly fixed day by day.

**Operation,** February 11. The still open mastoid wound was filled with gauze and the auricle freed by an incision above and in front, so that it could be turned downward. Flexible steel tourniquet applied, exerting its pressure along the lower edge of the middle fossa. A large osteoplastic flap, about three inches square, made. Lumbar puncture performed to allow the brain to fall back. A dural flap turned down, and the brain explored with a Cushing explorer. *The cortex punctured 1¼ inches above the external auditory meatus and the searcher carried inward horizontally to a line running from one external auditory meatus to the other. At a depth of 6½ cm. from the cerebral surface, the searcher encountered firm resistance, on overcoming which apparently clear fluid escaped under pressure. Mistaking the fluid for cerebro-spinal fluid from the ventricle, I slowly withdrew the searcher. A drop of thick pus followed along its track. Re-introduction of the searcher was followed by evacuation of from a half to three-quarters of an ounce of thick pus.* The effort to pass a thick instrument alongside the searcher for the purpose of facilitating the introduction of drainage simply resulted in forcing pus out of the searcher; and as more damage would be done by injury to the brain tissue in attempting permanent drainage, the searcher was withdrawn and dura, bone and skin tightly closed in layers.

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[42] The patient's family stated that she recently had been interested in white slave tales in the daily papers and in the discussions at meetings of the King's Daughters.

In a short time it was realized, however, that the failure to open the cavity of the abscess with a blunt instrument, at this great depth from the surface, was due to the presence of a firm, limiting membrane, which latter had been perforated with the semi-sharp searcher, but which was too dense to allow its perforation by a blunt instrument.

**Postoperative History.**—Following the operation the patient's condition greatly improved. The paralysis of the arm and leg partially disappeared; the mental state underwent marked improvement; the distressing sexual delusions entirely disappeared, but the delirium and some disorientation and severe headache continued.

March 1, seventeen days after evacuation, condition much the same. Complete paralysis of arm and leg. Mental state much clearer. Slight disorientation and delirium at night; fairly rational during the day. Complained of "horrid dreams" at night, but knew they were dreams. Knew nothing of brain operation and refused to consent to another, as she remembered the mastoid operation. Slight suppuration from upper edge of wound, thought to be due to stitch abscess.

March 19 (two months after primary trouble), the patient requested that something be done to relieve pain in the head.

**Examination,** March 20: "Left lower extremity slightly spastic; a moderate Babinski, and a few clonic twitches on testing Achilles reflex. Left knee jerk active; right knee jerk active. No Babinski; slight clonus. Left upper extremity, wrist, supinator and triceps jerks double plus; spasticity most marked distally. Gross movements in shoulder and elbow fairly good. Loss of passive motion in left upper extremity. Hemianesthesia most marked in upper extremity. Left abdominal reflexes diminished. Right very active."

**Mental Status.**—The patient is generally active and bright, and talks freely and readily. There is a moderate degree of psychomotor restlessness, and occasional delirium. Patient is well orientated as to place, but says: "I have not the least idea of the time of the day. I haven't seen a newspaper for several days to keep track of things. This must be about supper time; it is towards midnight." When told that it was 10:30 A. M., she replied : "I have lost track of things these last few days." She had a good insight into her condition, and gave no evidences of hallucinations, or other sensory perversions. Says, "I had another hand—I don't know what became of it. This one has no grip at all. I have lost the grip in my left hand. I have spent lots of time and energy trying to get it back. It is worth it to get the use of your hand." Keeps fumbling and searching with right hand over bed for the limb she imagined she had lost. Memory good for recent events.

**Previous history** showed partial disorientation in space and time, but not as to person. The space orientation is most affected.

**Dreams,** pleasant and disagreeable (both). Not lengthy. For example, "Last winter in a snow storm, walking up a little hill there was a white casket filled with white flowers and this casket began to roll down the hill. I don't know what started it—whether there was a body in it, I do not know. It was simply a dream, a disagreeable one." (Dr. C. Beling.)

**Conclusions.**—Hemianesthesia, hemiplegia, and moderate delirium (toxic).

**Operation,** March 21. The osteoplastic and dural flap again turned down, disclosing an area of granulations, which apparently follows the original exploration tract into the cerebral substance and communicates externally with the supposed stitch abscess. Following with a searcher the granulating tract inwards, at a depth of 6½ cm. from the surface, a dense capsule was again encountered; on perforating this the searcher entered the abscess cavity. Through the searcher the cavity was now completely evacuated and irrigated by alternately filling with normal salt solution and emptying with a suction apparatus. A Whiting encephaloscope, which is but 4½ cm. in length, was then introduced as deeply as possible over the searcher; the opening in the capsule made by the searcher was enlarged by the introduction and expansion of the blade of nasal alligator forceps, and through the opening thus made a rubber drainage tube, nine centimeters long, was introduced. The lower end of the tube lay in the cavity of the abscess, and the outer end was sutured to the skin through an opening rongeured out of the osteoplastic flap, the skin portion of the flap being horizontally stripped for that purpose. All the rest of the wound now tightly closed in layers.

March 22, the day following the operation, temperature 101 degrees. March 25, three days later, the condition was excellent; temperature normal. Involuntary movements of bowels at times. Still occasional delusions. Garrulousness much less. March 28, able to move arm and hand, to her great delight. Apparently normal mentally during the day, but had slight delirium at night.

April 11, paralysis of both arm and leg greatly improved. Hemianopsia greatly improved; with rough test with hand, apparently normal. Mental condition apparently normal.

June 29, still walked with cane. Used hand and arm almost normally. Mental condition absolutely normal. Had no recollection whatever of occurrences between the sinus operation and the first evacuation of the abscess. No memory of delusions, but remembered occurrences subsequently to first evacuation of the cerebral abscess.

November 15, mental condition normal. Remembered delusions and dreams well. No anesthesia left side. Walked with slight hemiplegic gait; used cane, but could go without. Used arm as well as ever. Slight spontaneous nystagmus in both corners, especially toward the affected side. Low voice heard at one foot with noise apparatus. Cold caloric test gave increased nystagmus, nausea and dizziness.

**Remarks.**—The case is of importance: first, because of its undoubted thrombotic *origin,* the abscess having been caused by vascular occlusion, as was evidenced by the "apoplectic" character of the onset of the paralysis, and not by direct extension of the suppurative process from the ear; secondly, because of the *unusual psychic symptoms* present, to which little attention has been paid in brain abscess; thirdly, because of its deep position, it being, so far as the writer knows, the only abscess of the internal capsule which has been successfully dealt with surgically.[43]

In cases of abscess of the brain from *direct extension* from contiguous suppurative lesions, paralysis, when present, is evidenced first by paresis of a part, generally the arm, then of the leg, and ultimately, with the development of the abscess, a complete paralysis ensues. In no record is to be found the history of a sudden paralysis, such as is common in apoplexy.

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[43] MacEwen: Pyogenic Diseases of the Brain and Spinal Cord; 1893, p. 152. (Makes the statement that "There are no cases recorded in which there is undoubted evidence of secondary involvement of the internal capsule from abscess of the temporo-sphenoidal lobe").

In the case here reported it was the sudden onset of the paralysis which led to the diagnosis of a vascular lesion, the probability of which was further strengthened by the age of the patient. However, a low blood pressure and the absence of a heart lesion, associated with an infection that has been sufficiently virulent to cause a perisinus abscess with disease of the sinus wall and complete suspension of the labyrinthine function was evidence that such a virulent infection *might* have caused a suppurative thrombus which at last found lodgment in the central ganglia. It is possible that this thrombus was thrown off twenty-four or thirty-six hours before its lodgment in the brain, or what is more probable, that it was a retrograde embolism causing *vascular* occlusion of one of the large deep veins of the brain.

It may be said, on the other hand, that the internal capsule was never directly involved; that the abscess simply pressed against it, and much can be said in favor of this latter supposition. However, the sudden onset of the paralysis, the location at which the abscess was found, exactly at the depth, from the cerebral surface, of the internal capsule, would seem to support the former contention.

Anatomical studies before operation have demonstrated that the internal capsule is situated, in the average brain, one and one-fourth inches above the centre of the external auditory meatus, and six centimeters inwards, on the line toward the opposite side. It was this exact anatomical position that made successful exploration possible at such a depth for such a small cavity.

We must assume, theoretically, from this case, that septic thrombus involving the internal capsule, with subsequent abscess, does not cause a permanent paralysis if drained.

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# CHAPTER VI.

# CEREBELLAR ABSCESS.

## SECTION I.

## Origin—Surgical Anatomy—Classification—Surgical Considerations.

### ORIGIN.

The vast majority of cerebellar abscesses originate from aural infection. In 117 recorded post-mortems analyzed by the author, the abscess was of otitic origin in 99 cases, from sphenoid sinus suppuration in 2, metastatic in 2, traumatic in 2, tuberculous in 2, carcinomatous in 1, and syphilitic in 1.

**Immediate Occasion.**—In adjacent cerebellar abscess, while acute exacerbation of a chronic suppuration is generally the immediate cause, trauma plays an important part. Many cases are recorded of cerebral symptoms immediately following a radical mastoid operation, or during the dressing of the wound, the inference being that the cerebellar abscess was either the direct result of the surgical manipulation, or was occasioned by secondary infection during the dressing.

Operative manipulation apparently induces different types of intradural suppuration, dependent upon the acuteness or chronicity of the suppurative bone lesion. Operations during acute mastoiditis, which occasionally induce a suppurative meningitis, apparently do not cause cerebellar abscess, without at least the intermediary of a sinus thrombosis. In chronic suppurative otitis, however, operative manipulation, or secondary infection may originate a retrograde infective thrombosis within the vessels of the bone from which the infection may be carried into the cerebral tissue itself.

### SURGICAL ANATOMY OF THE POSTERIOR FOSSA.

The descending portion of the lateral sinus divides the intradural contents of the posterior fossa into two unequal portions: (1) The *anterior one-third,* inclosed within the inaccessible triangle of the petrous pyramid, the basilar portion of the occipital bone in front, and the lateral sinus behind, contains the anterior one-third of the cerebellum, the pons and most of the medulla; (2) the *posterior two-thirds,* contains the lateral lobes of the cerebellum and the posterior portion of the medulla.

The more forward the sinus the greater the surgical inaccessibility to the contents of the anterior triangle of the posterior fossa.

## SURGICAL CLASSIFICATION OF ADJACENT CEREBELLAR ABSCESS.

From an operative standpoint, therefore, cerebellar abscess should be divided into two general classes, according to its location: (I) cerebellar abscess situated *on or near the anterior surface* of the cerebellum, the infection entering through the posterior surface of the petrous pyramid; (II) cerebellar abscess situated *in or on the posterior two-thirds* of a lateral hemisphere, the infection originating in the lateral sinus.

An abscess in either location, according to its position and origin, may be: (1) *meningeal,* limited by meshes of the piarachnoid and the inner surface of the dura; the suppuration resulting from an adjacent infective process by direct extension; (2) *intracerebellar,* without macroscopical evidence of cortical involvement; originating from retrograde thrombophlebitis, perivasculitis or metastatic vascular occlusion; and (3) *intracerebellar,* with a cortical "stalk."

### SURGICAL CONSIDERATIONS.

In every case of suspected cerebellar abscess, before exploration within the dura, the following physiological and pathological factors should be considered:

(1) The small size of the cerebellar fossa and the position of the lateral sinus, which render difficult an exposure through a clean area of sufficient size to permit of inspection and complete evacuation.

(2) The *increase in the bulk* of the affected cerebellar *hemisphere,* and *its displacement* with the contiguous brain stem beyond the median line, and with possible herniation of the brain stem into the foramen magnum.

(3) The cerebellar displacement which may obstruct the cerebro-spinal system circulation, causing acute obstructive internal hydrocephalus.

(4) The expulsive and uncontrollable herniation resulting from

the increased brain bulk, and disturbance of the cerebro-spinal circulation system.

(5) The possible immediate fatal effect of further herniation or displacement.

(6) The location of the abscess in or on the different lobes and surfaces of the cerebellum, dependent upon the path by which the infection enters the dura.

To meet these factors successfully the operation must be planned:

(1) to evacuate the abscess at its nearest approach to the dura, so that the operative manipulations will cause the least possible damage to the cerebral tissue; (2) to reduce the increased intracranial pressure; (3) to prevent excessive cerebellar herniation which, otherwise, may cause rupture of the piarachnoid, injury to the cerebellar tissue, and sudden dislocation of the brain stem, already abnormally displaced.

### HERNIATION IN EXPLORATION FOR CEREBELLAR ABSCESS: ANATOMICAL AND PHYSIOLOGICAL FACTORS.

The tegmen cerebelli rises dome-like (MacEwen's Atlas,[1]) from the superior border of the petrous pyramid, the upper limit of the dome reaching the plane of the floor of the anterior fossa, the third ventricle, and the central ganglia, and rising above the lower level of the descending horns of the lateral ventricle (MacEwen's Atlas, Fig. 49). Externally, the plane of the upper limit of the cerebellar dome is marked by a line extending from the level of the roof of the orbit to the upper border of the auricle.

In cerebellar abscess from infection of the labyrinth, a large part of the cerebellar substance is above the plane of the primary infection, viz., the posterior surface of the petrous pyramid.

The *intracerebellar* pressure is largely influenced by the cerebrospinal circulatory system, the fluid passing through the aqueduct of Sylvius into the fourth ventricle with its communications with the basal and lateral cisterna, situated near or on the anterior and inferior surfaces of the cerebellum. The fluid acts as a water-bed for the brain, lifting the brain stem and cerebellum upwards. The maximum intracerebellar pressure is exerted normally backward and outward, in the axis of the lateral lobes of the cerebellum, and upward nd outward, toward the tegmen cerebelli, away from the median ne and the internal portion of the posterior surface of the petrous yramid.

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[1] MacEwen, William: "Atlas of Head Sections," Glasgow, 1893.

### PATHOLOGICAL FACTORS.

Increase in cerebellar bulk, displacement beyond the median line, and obstruction to the cerebro-spinal system circulation, are the pathological factors to be considered. Intracranial pressure is enormously increased. This increased pressure is the result of the mechanical increase in the cerebellar contents from the abscess; an associated increase in the amount of the cerebro-spinal fluid from irritation of the inflammatory process; and the mechanical displacement, by the abscess, of the affected lobe beyond the median line into the cerebellar fossa of the opposite side, blocking the normal cerebro-spinal fluid pathway and causing an obstructive internal hydrocephalus. (See Internal Hydrocephalus, appendix to Cerebellar Abscess.)

When a hemisphere is the seat of an abscess its bulk is greatly increased; it gradually encroaches on the opposite cerebellar fossa, displacing the mid-line of the cerebellum and brain stem, causing compression of the opposite hemisphere. The cerebro-spinal circulatory system, in its effort to protect the vital centres by lifting the brain, presses the lateral lobes firmly against the dura—the place of maximum intracerebellar pressure—in the line of the dural incision.

The cerebro-spinal fluid, passing through the aqueduct of Sylvius and into the fourth ventricle with lateral branches to the basal cisterna, exerts a direct pressure on the brain, forcing it backward and upward. If now the displacement of the affected hemisphere is sufficient to obstruct the small opening for the third ventricle into the fourth, an acute obstructive internal hydrocephalus will result at once, adding enormously to the already increased intracranial pressure, and causing herniation of the brain stem into the foramen magnum.

### SURGICAL IMPORTANCE OF DIRECTION OF MAXIMUM INTRACEREBELLAR PRESSURE.

During increased intracerebellar pressure, the direction of the maximum pressure is unchanged, viz., upward and outward, away from the third and fourth ventricles, and the basal and lateral cisterna. As a result of this increased pressure the cerebellum itself is forcibly crowded backward and outward against the occipital bone, and upward and outward against the tentorium cerebelli (See MacEwen's Atlas, Fig. 42). This direction of maximum pressure is maintained until the increase of the intracranial pressure becomes so great that the normal resistance to displacement downward is overcome, when the medulla is herniated into the foramen magnum, with

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sudden death from pressure upon the vital centres,—a frequent termination of cerebellar abscess.

It is the direction of the maximum pressure that explains the long recognized operative observation, that in cerebellar abscess incision of the dura over the lateral lobe is frequently associated with uncontrollable herniation, while incision of the dural covering of the anterior surface of the cerebellum behind the posterior surface of the petrous pyramid is unattended by herniation. In cerebellar abscess of the anterior third, if the dural incision is over the lateral lobe, the herniation and the operative injury to the mass of cerebellar tissue intervening between the abscess and the dural opening, will add unnecessarily to the shock of the operation, and prevent complete primary evacuation or subsequent drainage of the abscess cavity (Figs. 25 and 26).

## ROUTES OF INFECTION.

### SURGICAL IMPORTANCE OF DETERMINING THE PROBABLE PATH OF INFECTION, WHETHER THROUGH THE PETROUS PYRAMID OR THE LATERAL SINUS.

Before operating the surgeon should endeavor to ascertain the probable path of infection of the suppuration, for on it generally depends the position of the abscess in (1) the anterior one-third of the cerebellum, or (2) the posterior two-thirds. The probable position of the abscess will largely determine the operative method to be adopted.

The importance of approaching the abscess at its nearest point to the surface was exemplified in a case in which, having exposed posterior to the sinus, because of a functionating static labyrinth, the herniation of the brain through the dural opening, in spite of ventricular puncture, made it impossible to depress the cerebellum far enough toward the median line to allow inspection of its anterior surface. Exploration *through* the cerebellar tissue located an abscess at a considerable depth from the dural opening. At the post-mortem the abscess was found situated within the meshes of the piarachnoid entirely anterior to the cerebellar substance. In such cases complete evacuation anterior to the lateral sinus (following the path of infection and using the walled-off piarachnoid) should be followed by a high percentage of recoveries.

### INFLUENCE OF PATH OF PRIMARY INFECTION ON SITUATION OF ABSCESSES.

Examination of the post-mortem reports of cerebellar abscess shows that in a general way the location of the abscess in or on the

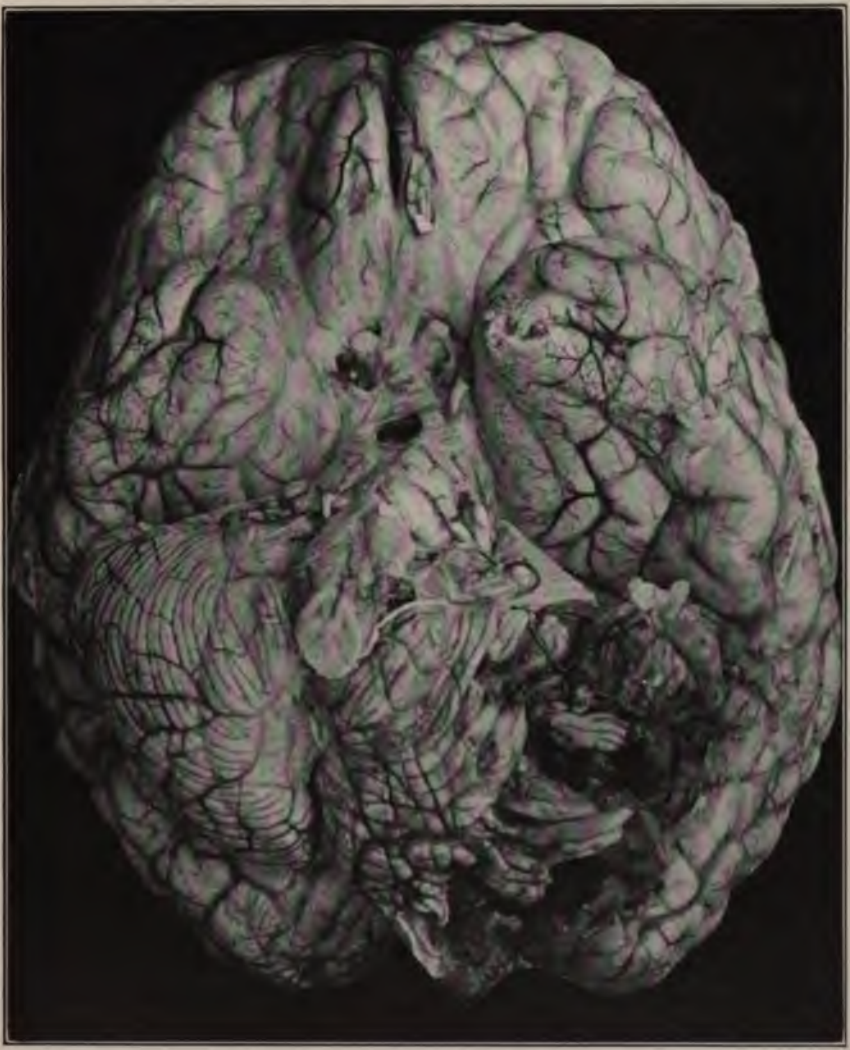

FIG. 26.—Intrapiarachnoid abscess situated on the anterior surface of the cerebellum. Technical error was made of exposing only one hemisphere, and this behind the lateral sinus, necessitating exploration and evacuation of abscess through the substance of the cerebellum, followed by extensive brain herniation and necrosis; whereas, if the sinus had been obliterated, the abscess could have been evacuated without cerebral injury. A part of the normal cerebellar tissue was torn away in removing the brain, after death, from the dura. Note the displacement of the brain stem and the displacement of the diseased hemisphere beyond the median line.

cerebellum is dependent upon the path of extension into the cerebellum from the original site of infection. Determination of the route of infection, then, becomes of the greatest surgical importance; and as experience teaches that exploration and drainage through the original route of infection results in the largest proportion of recoveries in cerebellar abscess, the diagnosis of the exact route of infection into the cerebellum will largely determine the operative procedure.

The routes of infection in the cerebellar abscesses are: (1) around or through the *labyrinth*; (a) through caries of the cells within the bony capsule of the petrous pyramid—the cancellous tissues surrounding the bony semi-circular canals and cochlea; (b) through caries or necrosis of the capsule of the semi-circular canals; (c) from suppurative labyrinthitis by "pre-formed ways," namely, the ductus endolymphaticus, the internal auditory meatus, or the aqueductus cochleæ;[2] (2) through thrombosis of the lateral sinus and its communicating veins.

An examination of the records of post-mortems of cerebellar abscess reported in the literature shows the path of infection to the cerebellum in only 128 cases. So far as the writer is aware no systematic research from post-mortem records has previously been made to ascertain the relative frequency of the individual paths of infection into the cerebellum.

ROUTES OF INVASION TO CEREBELLUM IN 125 CASES[3]

| *Labyrinth:* | | | *No. of Cases.* | *Percent.* |
|---|---|---|---|---|
| Labyrinth | | 19 | 56 | 44.8 |
| Semicircular canals | | 6 | | |
| Vestibular aqueduct | 11 | 17 | | |
| Retrograde thrombosis from empyema of the vestibular aqueduct | 1 | | | |
| Ductus endolymphaticus or sacculus endolymphaticus | 5 | | | |
| Internal auditory meatus | 7 | 8 | | |
| Facial nerve | 1 | | | |
| Subarcuate hiatus | 4 | 5 | | |
| Fold passing from dura to mastoid cells, probably subarcuate hiatus | 1 | | | |
| Undetermined, but thought to be by "pre-formed way" | 1 | 1 | | |

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[2] Mackenzie, G. W.: Labyrinth Papers; Philadelphia, 1913, 222, p. 8.

[3] Eagleton, Wells P.: Cerebellar Abscess; *Jour. Amer. Med. Assoc.*, Oct. 4, 1919, Vol. 73, pp. 1060-1062.

| | | *No. of Cases.* | *Percent.* |
|---|---|---|---|
| Total Forward, | | 56 | 44.8 |
| *Caries of Petrous Bone* | | 22 | 17.6 |
| (There can be no doubt that many of these were labyrinth cases.) | | | |
| *Sinus Thrombosis:* | | 41 | 32.8 |
| (Associated sinus thrombosis and labyrinthitis).<br>Of 97, the total number of cases of infection from the labyrinth and sinus thrombosis, 13 (or 13.3 per cent.) had both labyrinthitis and sinus thrombosis; showing the intimate association of the blood stream infection with cerebellar abscess. Cerebellar abscess with labyrinthitis need not necessarily be situated in the anterior one-third of the cerebellum, as an associated sinus thrombosis may be the immediate origin of the abscess. | | | |
| *Necrosis of Mastoid* | | 3 | 2.4 |
| *Metastatic* | | 1 | .8 |
| (From Pulmonary Suppuration.) | | | |
| *Unclassified:* | | | |
| Fistula of Dura from Extradural Abscess, probably Perisinus from description | 1 | 2 | 1.6 |
| Fistula of Neck | 1 | | |
| | | 125 | 100.0 |

# CEREBELLAR ABSCESS.

## SECTION II.

### In the Anterior Portion of the Cerebellum Originating from Infection of the Petrous Pyramid.

### ORIGIN.

The labyrinth is so frequently the cause of cerebellar abscess that the first step towards localization in a suspected brain abscess is the investigation of its function. As sensory aphasia, not of vascular origin, but accompanied by a left-sided dural suppuration, is strongly suggestive of temporo-sphenoidal abscess, so a dead labyrinth, associated with symptoms of brain abscess, presumably localizes the lesion within the posterior fossa. On several occasions I have explored for and evacuated a cerebellar abscess in patients brought to the hospital in coma, regarding whom no previous history was obtainable at the time of operation, the diagnosis resting solely on a chronic suppurating otitis, evidences of cerebral compression, and a non-reacting static labyrinth.

### INFECTION OF CEREBELLUM FROM BONY CARIES OR NECROSIS.

The probability of the infection extending to the cerebellum depends largely on the anatomical structure of the individual petrous bone involved, *i.e.,* the presence or absence of perilabyrinth cells—the small cancellous cells within the bony capsule of the petrous pyramid,—especially along the inferior and posterior surfaces.

From operative findings I would place the path of infection from the labyrinth to the cerebellum in the following order of frequency. (See Routes of Infection, p. 88):

(1) By way of the cells surrounding the bony semicircular canals and cochlea. These can be subdivided into two groups, through which infection occurs: (a) the cells surrounding the compact bony labyrinth (perilabyrinth cells) in the posterior portion of the petrous pyramid; (b) the cells in Trautmann's triangle, the outermost of the posterior perilabyrinth cells.

(2) Through nutritional necrosis and caries of the bony labyrinth capsule itself.

When the infection goes around, not through the labyrinth, the cells of Trautmann's triangle are affected; but they are not as generally the *direct* cause of infection of the cerebellum as is supposed. The deeper cells, however, (from which the direct point of entrance of the infection to the brain occurs) are involved through the cells in Trautmann's triangle.

The reports of the post-mortem examinations[4] of cerebellar abscess show that the dura is frequently adherent over the posterior surface of the petrous pyramid, associated in a small proportion of cases with a fistulous opening marking the site of the entrance of the suppuration into the cerebral substance.

CASE XVIII.

F. C.: Intrapiarachnoid Abscess. Girl, aged eight years. Hypertrophy of adenoids and tonsils, which had been imperfectly removed. Tuberculosis of left lung. Hematuria. Chronic otitis media, left ear, with discharge, purulent and in large amount. Adenoids and tonsils removed, and mastoid operation. Since then had had several hemorrhages from kidneys and had been passing little urine. Had vomited considerably.

**Symptoms.**—Spontaneous nystagmus, vomiting, mild papillœdema in both eyes of three weeks duration. Vomiting originally thought by attending physician to be due to kidney lesion. Thought to hear well, but the peevish, restless condition of child made this unsatisfactory. Urine contained pus, blood and albumen; specific gravity 1026. Weber referred to left. Thorough examination impossible because of child's peevish condition. Chronic nephritis. Symptoms now pointed to cerebellar abscess.

**Operation.**—In spite of the suppression of urine ether was administered and the patient placed in left lateral position on operating table. When under anesthetic she was turned on right side and the head placed in a movable forward carrier. She was then rotated on the chest with sandbags under each shoulder. Incision in left side, extending from mastoid to median line and from median line downward about three inches; the flap was rapidly turned outward. Difficulty encountered in controlling hemorrhage from the large accessory veins.

The dura being very tense an opening was made over the occipital lobe and the searcher passed in an attempt to find the ventricle. The first passage of the searcher failed to reach the ventricle after passing to a depth of six centimeters. The direction probably was too far outward. The second passage apparently was straight forward toward the nose. A large amount of clear cerebrospinal fluid was evacuated. On withdrawal of the trocar more fluid followed. Closure of dura over ventricular puncture opening.

Immediately after ventricular puncture the dura over the cerebellum became much softer. Dura incised. Immediately herniation through large dural opening prevented inspection. Searcher passed through substance of cerebellum toward posterior surface of petrous portion of temporal bone at a depth of four centimeters from the cortex. Thick pus evacuated, caught by suction apparatus. Pus ran slowly on account of its thick character, being first muco-pus followed by pure pus. No capsule was felt during passage of searcher, although a capsule was expected because from the history cerebellar symptoms had lasted from three to four weeks. Introduction of drain. Death two days later.

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[4] For Analysis of Post-Mortem Records, see "Condition of Dura over Site of Infection." Appendix II, Cerebellar Abscess.

**Autopsy Findings.**—On reflecting the dura the brain well filled the cranial cavity and there was exposed a very light, grayish-white brain substance. On the left side of the posterior fossa was an opening in the dura opposite to the trephined opening, about one inch in diameter, and a small amount of softened, purulent brain matter was found clinging to the dura over the posterior surface of the petrous portion of the left temporal bone. The base of the skull after removal of the dura was everywhere clear and free except in the following places:—in the posterior fossa on the left side, over the posterior surface of the petrous portion of the left temporal bone, where there was an oval, reddened, suppurating mass about one centimeter in diameter, which was firmly adherent to the dura. On tearing off the dura from the bone there was exposed an oval area of bone necrosed about one centimeter in diameter on the posterior surface of the petrous portion of the left temporal bone. The dura on the inner surface was smooth and glistening over this area of necrosed bone. The cortex of the bone around this area was entirely gone, leaving a pale, reddened granular tissue beneath. The rest of the left half of the posterior fossa showed the two openings and was quite clear.

On removing the brain there was seen in the middle fossa a considerable amount of fluid, slightly cloudy, and what appeared to be a purulent exudate around the region of the pituitary body and the tips of the temporo-sphenoidal lobes. (See Fig. 26.)

The anterior and middle fossæ and the posterior fossa on the right side were everywhere clear and free. In the foramen magnum there was a small amount of purulent exudate around the upper part of the cord. The ethmoid cells were slightly moist, but everywhere clear and free from suppuration. The inferior surface of the pituitary was clear and free. The sphenoid cells showed a small amount of mucoid material, but were free from suppuration. Both lateral sinuses were clear and free.

**Comment.**—In spite of the hemorrhage and the weak condition of the patient, the cerebellum over both hemispheres and forward to the sinus should have been exposed, as it is impossible to inspect both cerebellar hemispheres through an opening over one of them. The patient had been suffering from symptoms of labyrinthine irritation for at least three weeks. In view of the fact that there was negative blood culture, the sinus should have been ligated and the abscess evacuated in front and through the sinus. The abscess was entirely intrapiarachnoid.

The operative demonstration of a fistulous opening, or of adhesions between the dura and cerebellum, is of the same surgical importance as similar lesions on the roof of the antrum and middle ear, and should in like manner influence the treatment.

Intrapiarachnoid abscesses are most frequent on the posterior surface of the petrous pyramid near the apex, as it is here that all the cellular tracts of the labyrinthine cells coalesce.[5] This also accounts for a large number of intra-cerebellar abscesses which at operation have been found located deep within the cerebellum, near the median line.

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[5] Girard, L.: Perilabyrinthine Cells: *Société de Laryngol., d'Otol. et de Rhinol. de Paris*, December 9, 1911; Also, *International Centralblatt für Ohrenheilkunde*, 1912, B. X., p. 156.

In the selection of the proper approach to the cerebellum a functionating labyrinth does not necessarily exclude the posterior surface of the petrous pyramid, as with infection of the peri-labyrinthine cells the labyrinth itself may not be involved. A carefully taken history, however, will always reveal a previous "irritation" of the labyrinth.

Hepe[6] reports a case of deep-seated epidural abscess, without labyrinthitis.

> The histological findings cleared up the genesis of the leptomeningitis; it originated through the rupture of a deep-seated epidural abscess derived from the pneumatic cells.

### Frequency of Different Forms of Labyrinthine Suppuration Causing Cerebellar Suppuration.

Cerebellar suppuration may follow any one of the four types of intralabyrinthine suppuration, namely, (a) suppurative labyrinthitis with closed empyema within the bony labyrinthine cavity, the vestibule, the semi-circular canals and cochlea; (b) caries of the compact bony walls of the semi-circular canals, vestibule, or cochlea, associated with secondary circumscribed or diffused labyrinthitis with granulation tissue replacement of the membranous labyrinth; (c) necrosis of the petrous pyramid from nutritional disturbance with secondary labyrinthitis; and (d) infective labyrinthitis through "preformed ways," the ductus endolymphaticus, aqueduct cochleæ, along the perineural sheaths of the internal auditory meatus (West),[7] or between the layers of dura of the saccus endolymphaticus (Horne).[8]

Cerebellar abscess, in the author's experience, most frequently follows caries of the capsule of the posterior semi-circular canals. At operation, the compact bony wall on the posterior surface of the petrous pyramid is found to be perforated. This process is generally associated with obliteration and absorption of the other semi-circular canals.

Closed empyema of the labyrinthine cavities frequently causes intrapiarachnoid suppuration in the neighborhood of the internal auditory meatus, but rarely intracerebellar suppuration.

Labyrinthine necrosis with the formation of a sequestrum as a result of nutritional death—a condition frequently seen in tuberculosis, the sequestra often involving the whole petrous pyramid,—

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[6] Hepe: Ein Beitrag zur Kasuistik der tiefgelegenen epiduralen Abszesse ohne Labryinthentzündung; *Zeitschrift für Ohrenheilkunde*, 1916, Bd. 74, S. 91.

[7] West and Scott: Operative Surgery of the Labyrinth; *Proceedings Royal Medical Society*, April, 1908, p. 918.

[8] Horne, Jobson: Formation of a Circumscribed Intradural Abscess at the Site of the Saccus Endolymphaticus; *Transactions Sixth International Otological Congress*, London, August, 1899, p. 393.

is but rarely associated with cerebellar abscess, though it frequently causes death from meningitis.

In acute infective labyrinthitis the infection may extend along the perineural arachnoid sheaths of the nerves of the internal auditory canal, distending them with turbid or hemorrhagic fluid.

### ABSCESS IN LATERAL CISTERNA.

Acute suppuration extending through the internal auditory meatus along the perineural arachnoid sheaths of the facial and auditory nerves, while usually causing diffuse suppurative meningitis, is not infrequently followed by a circumscribed collection of purulent cerebro-spinal fluid in the lateral cisterna.

### SECONDARY SEROUS LABYRINTHITIS.

From the proximity of the caries of the deep perilabyrinth cells to the delicate structure of the labyrinth, there is generally an associated serous labyrinthitis, as evidenced clinically by a non-reacting static labyrinth.

Abscess secondary to sinus involvement, although associated with petrous pyramid infection, is not necessarily situated in the anterior one-third, or adjacent to the petrous pyramid, but may be in any part of the cerebellum.

**Clinical Classification.**—*Abscesses* of the following clinical varieties may be *situated in part or wholly in the anterior one-third of the cerebellum:* (1) *Adjacent, Meningeal,* chronic or acute, limited by the meshes of the piarachnoid and the inner surface of the dura: the intradural suppuration resulting from an adjacent infective process by direct extension from infection of the petrous pyramid. (2) *Adjacent, intracerebellar, without macroscopical evidence of cortical involvement;* chronic with capsule, or acute without limiting membrane. (3) *Adjacent intracerebellar with capsule* or "stalk."

**Principles of Surgical Treatment.**—Physiological and mechanical factors and clinical experience require that the exploration of a cerebellar abscess, *originating from infection of the petrous pyramid,* be made through a dural opening, situated as far forward as possible in order to follow the route of infection, and to lessen the cerebral herniation. The dural opening must also be large enough to permit of adequate inspection and evacuation, and it must be made through a clean field. (See Surgical Factors, p. 85.)

**Operative Approach to the Cerebellum.**—Two routes of approach to the cerebellum are followed by surgeons, the choice resting largely upon the operator's attempt to explore through a clean area, or to follow the path of infection.

In exploration through a clean area the method involves:

*A. Exposure of one or both lateral lobes of the cerebellum, with exploration and drainage behind the lateral sinus.*

Exposure of one lateral lobe and incision of the dura through the opening is fundamentally wrong, as the lateral displacement of the affected lobe beyond the median line causes immediate uncontrollable herniation which prevents all inspection. Numbers of cases of abscess, however, have been successfully explored and drained by this method.

*B. Incision and evacuation in front of lateral sinus.*

The route anterior to the sinus is especially advantageous if the abscess is located in the meshes of the piarachnoid, in contact with the petrous pyramid. In this case the abscess is united by protective adhesions to the dura over the petrous pyramid; evacuation and drainage in front of the sinus follow the path of infection, and permit of preservation of the protective adhesions, while the abscess is approached at its most superficial point.

Exploration in front of the sinus, however, presents the following disadvantages: (1) if the abscess is not found, meningitis is almost sure to follow, as the operation is conducted through the infected area of the mastoid; (2) the area between the sinus and the posterior pyramid is too small to allow of proper exploration, complete evacuation, or attempt at eradication.

The supposition that an abscess which originates from the labyrinth or petrous pyramid is located in the anterior one-third of the cerebellum, and consequently may always be reached in front of the sinus, is not borne out by post-mortems. Intracerebellar abscesses, situated in the substance of the cerebellum itself, are not uniformly near enough to the posterior surface of the petrous pyramid to be successfully explored and completely evacuated through the small space in front of the sinus. A large number of unsuccessful explorations are recorded. Intrapiarachnoid abscesses originating from the posterior surface of the petrous pyramid are connected with the original focus of infection. However, they may extend great distances either upward under the tentorium, or downward along the under surface of the cerebellum.

*C. Destruction of the labyrinth followed by exploration of the cerebellum in front of the sinus: Enlargement of the dural open-*

*ing following application of two closely allied encircling ligatures to the sinus and incision between them:* (Bourguet).[9]

While disturbance to the return circulation by obliteration of the lateral sinus is generally slight,[10, 11] the mechanical difficulties of occlusion by ligation of a pervious large sinus are so great that it has been practised hitherto in but two recorded instances.[9 and 12]

*D. Primary exposure of the lateral sinus from behind; obliteration of its lumen,* above *by invulsion* of its outer wall into its cavity, and below by an encircling ligation; *followed by free incision through the walls of the sinus,* with enlargement of the incision anteriorly into the contiguous dura surfaces.

By this method the exposure and exploration may be conducted through a clean field, away from, although adjacent to the primary site of infection, the petrous pyramid. The dura is incised away from the plane of maximum intracerebral pressure, and consequently, the herniation is reduced to a minimum, and the junction of the anterior and lateral surfaces of the cerebellum is freely exposed.

If associated with ventricular puncture, this method allows of inspection of the anterior surface of the cerebellum—the situation of adjacent intrapiarachnoid abscesses of the posterior fossa, and of exploration of the anterior one-third of the cerebellum—the usual location of adjacent intracerebellar abscess from infection of the petrous pyramid. If an abscess is located, it provides ample room for its thorough inspection, cleansing and obliteration.

### Anatomy of the Lateral Sinus on Cross Section.

The lumen of the descending portion of the lateral sinus, opposite the middle ear, is an irregular oval with its greatest convexity outward, the sinus tunneling, as it were, between the layers of the dura (MacEwen's Atlas, Fig. 39).[13] The inner wall of the sinus is flush with the contiguous dura, while the outer wall projects considerably beyond the surface of the dura. Obliteration of the sinus by an encircling ligature is difficult, because in its horizontal diameter it presents *two* fixed points of dural surface that cannot be approximated. Reaching the knee, the sinus becomes distinctly pear-shaped, with its inner wall still flush with the contiguous dural surfaces of the cerebellum and cerebrum. The superior petrosal sinus enters at an oblique angle, emptying into the *inner* wall at its posterior margin. This is of surgical importance. After passing the knee, *the horizontal portion* of the lateral sinus assumes a triangular shape, with three fixed points, the dura of the tentorium cerebelli being interposed (MacEwen's Atlas, Fig. 10). Because of the three fixed points of dural attachment, to completely obliterate a large transverse sinus by an encircling ligature is difficult; the tightening of the ligature tears the dural attachments, with resulting hemorrhage.

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[9] Bourguet: Abscès du cervelet et labyrinthine suppurée; trépanation; guérison; *Toulouse Medical,* Tome 15, p. 289.

[10] Eagleton: Circulatory Disturbances Following Ligation of the Internal Jugular Vein in Sinus Thrombosis, with Report of a Case; *Archives Otology,* 1906, Vol. XXV, 2, p. 991.

[11] Stolz: Ueber die Unterbindung der Vena jugularis interna; *Correspondenz-Blatt für Schweizer Aerzte,* 1918, Bd. 48, No. 13, S. 415.

[12] Ballance: Personal Communication to Dr. Edward B. Dench.

[13] Mall, F. P.: On the Development of the Blood Vessels of the Brain in the Human Embryo; *Amer. Jour. of Anatomy,* 1905, Vol. IV, p. 1.

In both the transverse and the descending portions of the sinus, however, it is possible to *invulse* the outer wall into the cavity, thus completely obliterating the sinus.

### PHYSIOLOGICAL AND ANATOMICAL FACTORS. BLOOD PRESSURE WITHIN THE SINUS.

The blood pressure in the descending portion of the lateral sinus, normally but a few millimeters of mercury, is negative during each inspiration, the collapse of the sinus being frequently witnessed during its operative exposure. The volume of blood, although under low pressure, is large. This low or negative pressure permits the control of hemorrhage, following injury to the sinus wall, by bridging the gap, the lumen of the sinus remaining permeable. The gloved finger, a small piece of cotton, or the "postage stamp method" (in which a piece of fascia lata is placed over the rent, the fascia adhering to the injured sinus), controls the hemorrhage without stopping the current of blood within its lumen. The control of hemorrhage from the sinus by compression is more difficult because of the large volume of blood and the triangular shape of the sinus.

**Surgical Obliteration of the Lateral Sinus.**—A device[14] holds the two ends of suture apart so as not to compress the fixed dural attachment, while the descent of a metal obturator causes an invulsion of the outer wall of the sinus into its cavity, obliterating the lumen. The obliteration of the sinus frees the approach to the anterior triangle of the cerebellar fossa. Through the enlarged triangle, the anterior surface of the cerebellum (the most frequent site of cerebellar abscess) can be inspected. (Figs. 27, 28.)

**Technic of Obliteration of the Lateral Sinus by Invulsion of its Outer Wall into the Lumen.**—A small opening is made in the dura, on each side of the sinus, using a triangular dural knife or cystotome. A ligature is passed

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[14] Eagleton, W. P.: An Original Device for the Control of Hemorrhage from the Large Sinuses of the Brain, by Involution of the Outer Wall into the Lumen; *Medical Record*, February 15, 1919, p. 274.

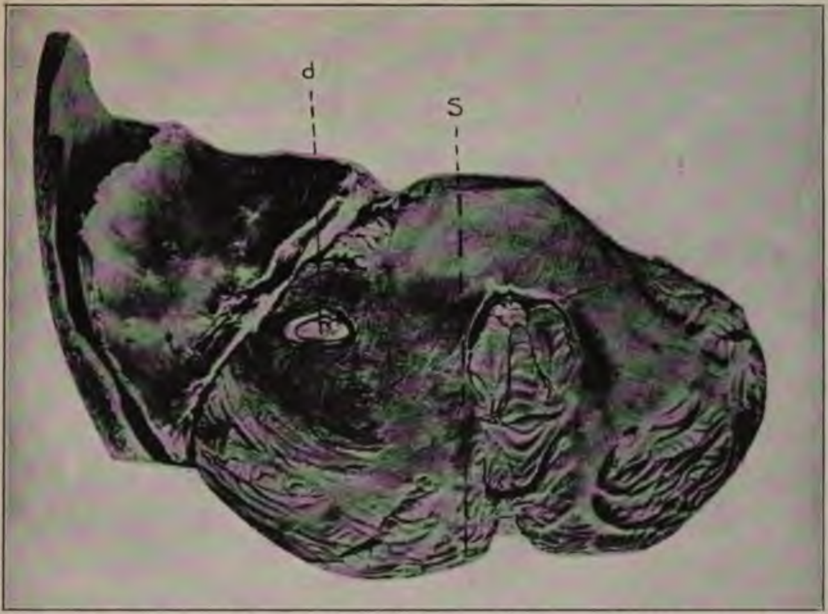

FIG. 25.—Temporal bone and cerebellum with abscess *in situ*, almost one-third of the affected hemisphere being displaced beyond the median line.

S—Sagittal plane; d—drain in evacuated abscess, with surrounding zone of encephalitis. (Copied from Hegener's Labyrinthitis und Hirnabszess; *Beiträge zur Anatomie, Physiologie, Pathologie und Therapie des Ohres, der Nase und des Halses*, 1901, Bd. 2, S. 461.)

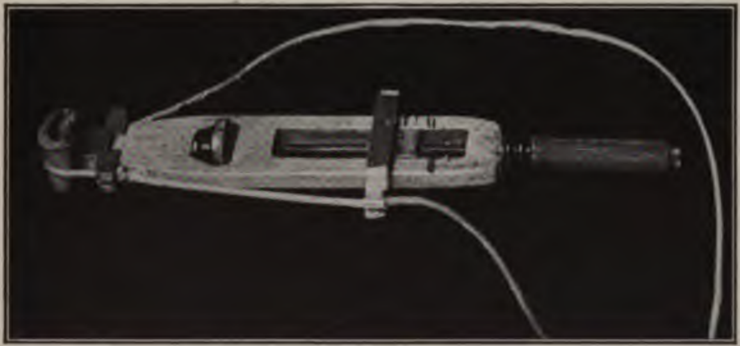

FIG. 27.—Author's sinus obliterator and carrier in position.

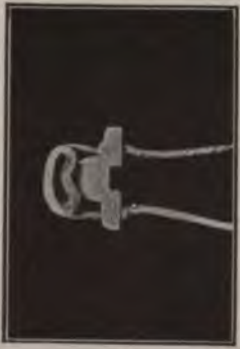

*FIG.* 28.—Obliterator detached from carrier.

from one dural opening to the other, using a full curved needle with the point blunted, damage to the cerebral tissue being avoided by hugging the dural surface. One side of the ligature is knotted at its centre and the suture placed in the slot of the carrier and of the obturator on the same side. The other side of the ligature is then placed in the opposite slot, using the knotted point of the ligature as a fulcrum, and tightened sufficiently to cause a slight indentation of the sinus, when the ends of the ligature are tied in a bow knot over the cross arm of the carrier. Tightening the upper screw of the carrier will cause the descent of the obturator into the sinus, obliterating its cavity. The slots in the soft metal arms of the obturator are pressed firmly against the suture holding it in position; the suture above is then loosened and removed from the slots of the carrier, and the obturator liberated from the carrier by unscrewing the lower screw. The suture is now tied over the obturator. If the ligation is to be made above the knee it will be necessary, in addition, to perforate the tentorium cerebelli with the needle. In passing the needle the entrance of the superior petrosal sinus into the lateral sinus may be injured. Hemorrhage from the petrosal sinus can be controlled by tucking under it a small piece of cotton with a black silk carrier.

The upper portion having been obliterated and all downward current of blood stopped, the lower portion of the sinus is easily ligated, by an encircling ligature, as the sinus walls are here compressible; or, if the bony exposure is low enough, the lower portion of the sinus may be obliterated by the pressure of a tampon against its firm bony wall. Between the two ligatures the sinus can be freely incised.

CASE XIX.

S. B.: Cerebellar Abscess.[18] Male, adult. Influenza followed by acute otitis media in right ear. *Mastoid operation performed*, neither dura nor sinus being exposed. Three weeks later wound had practically healed; no symptoms. Five weeks after operation, dull, boring pains commenced behind right eye and in right frontal region. Paroxysm of pain was followed by vomiting, and for two weeks there was headache of varying intensity on the right side and frequent projectile vomiting which was influenced by the position of the patient, who could take food when lying on the right side but would vomit upon turning on his back.

Two weeks later the patient was much improved and could walk about the room, although when he first attempted to walk he staggered and had a tendency to fall to the right. No strength in right arm; used left. Became dizzy on sudden change of position. Headache subsequently increased in severity, right-sided weakness became more marked, and the patient became slightly confused mentally. Examination showed the mastoid scar to be healed. No discharge from ear. Patient could hear with right ear. Cold caloric in right ear produced nystagmus, but not nearly so marked as that produced in left; complained of blurred and troubled vision. Ophthalmoscopic examination revealed congestion and slight œdema of both discs and fixation nystagmus. Mentality dull; no aphasia. Pulse 60; respiration irregular, bordering on Cheyne-Stokes. Cerebro-spinal fluid clear under pressure.

Movements of pronation and supination were overdone. Asynergia more marked on the right side, as shown in his inability to coördinate the various muscles and joints. Asked to put his right hand back of his head, his arm would apparently wobble in every direction until he got it in place, or he would raise the right hand with the left. The right hand was much weaker than the left.

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[18] Eagleton, W. P.: Report of a Successful Operation for Cerebellar Abscess by Obliteration of the Lateral Sinus and Exploration and Drainage through Its Inner Wall and Anterior Surface of Cerebellum; *Medical Record*, February 15, 1919, Vol. 95, p. 274.

**Second Operation.**—The old mastoid cavity was therefore re-opened and the sinus exposed, but no trace of infection was found.

**Third Operation.**—*Cerebellar:* Three days later, May 12, a cerebellar operation (W. P. E.) was performed, hemorrhage from scalp being controlled by von Hayden's instruments. The whole cerebellar fossa was exposed on the right side, and the posterior surface of the petrous pyramid removed as far as the internal auditory meatus. Difficulty was experienced in locating the sinus on account of granulations; but it was found by approaching from behind forward. A blunt needle threaded with heavy silk was passed just below the sinus knee encircling the dura from behind forward and out on the anterior wall of the cerebellar fossa. On first passing it the sinus was undoubtedly perforated. The sinus was obliterated by the instrument above at the knee, and was again tied with an encircling ligature as low down toward the bulb as possible. Between the ligatures the external wall of the sinus was then opened without loss of blood, and the incision was continued through the inner wall to the cerebellum. This incision was afterwards enlarged, the opening into the cerebellar fossa being directly at the angle of the anterior and external surfaces. There was no prolapse of the brain. Exploration with searcher at distance of 3 cm. located the abscess with capsule, pus flowing freely. On introduction of Whiting's instrument (encephaloscope), pus again flowed. The capsule could be felt at the bottom of the encephaloscope, and pressure expressed more pus. The abscess was thoroughly cleansed and wiped out. There was slight herniation of the brain. A rubber tissue drain was inserted and the wound closed. The obliterating obturator was removed on the tenth day. Uninterrupted recovery.

# CEREBELLAR ABSCESS.

## SECTION III.

### From Sinus Thrombosis Frequently Situated on One of the Surfaces in the Posterior Two-thirds of the Cerebellar Fossa.

#### IRREGULAR SHAPE OF CEREBELLAR ABSCESS; ITS SURGICAL IMPORTANCE.

Many charts depict cerebellar abscess as round or oval; postmortem examinations, on the contrary, show it frequently to be very irregular in shape, with prolongation from the main cavity, the opening into the prolongation at times being so small that it is frequently mistaken for an independent abscess cavity.[16]

The irregular shape of the cerebellar abscess is explainable by the recognized tendency of suppuration to follow white fibre tracts, avoiding the more freely nourished gray substance.

**Surgical Principles in Treatment of Adjacent Cerebellar Abscess.**—With adequate and properly selected exposure the principles outlined above may be applied as in the treatment of adjacent abscess of the middle fossa. In cerebellar abscess, complete evacuation or, if possible, eradication, is especially important; because with drainage alone the irregular shape of cerebellar abscess is frequently followed by refilling and extension of the suppuration. (See Secondary Abscess, p. 59.)

Attempts at eradication are necessarily attended with especial danger because of injury to the neighboring vital medullary centres during operation, or from subsequent compression.

### ABSCESS OF SINUS ORIGIN SITUATED IN OR ON POSTERIOR TWO-THIRDS OF CEREBELLUM.

Abscess in this location is secondary to sinus thrombosis and may result from retrograde thrombosis of a cerebellar vein entering the sinus,[17] or from necrosis of the sinus wall itself.

[16] Neumann, H.: Cerebellar Abscess; *English Edition*, 1907, p. 11.

[17] Some of the inferior cerebellar veins empty into the transverse sinus, while the sigmoid portion receives the internal auditory and some of the veins of the medulla and pons.

When secondary to a retrograde thrombosis the abscess is generally situated in the lateral lobe, and as the thrombosis is frequently very extensive any portion of the cerebellum may be involved in the abscess.[18, 19, 20]

When the cerebellar suppuration is secondary to a necrosis of the sinus, the abscess is *on* the lateral lobe—"superficial brain necrosis" from direct extension.

### I. CEREBELLAR ABSCESS WITH SECONDARY SINUS THROMBOSIS.

Cerebellar abscess may be the primary cause and not the result of a thrombosis of the lateral sinus.

With increased intracranial pressure the sinuses, on account of the low venous pressure, are early obliterated, and, according to Wolbach,[21] multiple small areas of brain tissue are herniated into the yielding points of the entrance of the arachnoid villi within the sinuses. The herniation is frequently followed by an occluding thrombus which, in the presence of infection, may undergo septic degeneration. The author has seen such an occlusion of the lateral sinus during an operation for cerebellar abscess. On opening the sinus no bleeding resulted, although no clot was present, the inner sinus wall being pressed firmly against the outer by the increased intracranial pressure of the abscess.

### II. INTERCURRENT (TERTIARY) ABSCESS SECONDARY TO SINUS THROMBOSIS.

Abscess secondary to sinus thrombosis may be: (a) *cortical* or *meningeal,* chronic or acute, from infection of the inner wall of the sinus, or of the lateral veins entering it. The situation may be on any surface adjacent to the infected sinus (Fig. 13); (b) *intracerebellar,* without macroscopical evidence of cortical involvement, with capsule, or without limiting membrane, from retrograde thrombophlebitis of the cerebellar veins. The situation may be in any part of the homolateral hemisphere.[22] Thirteen per cent. of all cases of cerebellar abscess with labyrinthine involvement are asso-

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[18] Neumann, H.: Der Otitische Kleinhirnabszess; 1907, S. 5.

[19] Ruttin, E.: A Clinical Study of the Serous and Purulent Abscesses of the Labyrinth; *Authorized translation by H. Newhart,* 1914, p. 183, Case 68.

[20] Martin, H. H., Savannah, Ga., and Crowe, S. J., Baltimore: Lateral Sinus Disease—Case Report; *Laryngoscope,* December, 1920, Vol. XXX, No. 12, p. 817.

[21] Wolbach, S. B.: Multiple Hernia of the Cerebrum and Cerebellum, Due to Intracranial Pressure; *Jour. Med. Res.,* Vol. XIX, No. 1; New Series July, 1900, Vol. XIV, p. 159. (Reports two cases of occluding thrombus in lateral sinus; Case I, associated wth acute otitis, Case VIII, unassociated, both just external to the torcular.)

[22] Jones, H. E.: Some Unmanageable Complications of Suppurative Middle Ear Disease, *Liverpool Med. Chir. Jour.,* 1899, Vol. XIX, p. 43; *British Medical Journal,* II, 1898, p. 1235; *Transactions International Otological Congress,* 6, London, 1899.

ciated with sinus thrombosis. The position of the abscess will thus depend upon whether it originated directly from the petrous pyramid or from the sinus.

### III. TRAUMATIC; ACUTE OR CHRONIC.

### IV. METASTATIC; ACUTE OR CHRONIC.

### TREATMENT OF ABSCESS IN OR ON THE POSTERIOR TWO-THIRDS OF THE CEREBELLUM FOLLOWING THROMBOSIS OF THE LATERAL SINUS AND ITS COMMUNICATING VEINS.

**Incision Through Inner Sinus Wall.**—If the diagnosis of cerebellar abscess is positive, and the abscess is secondary to a sinus thrombosis, and consequently is probably situated in the lateral lobe of the cerebellum, exploration and evacuation may be conducted through the inner wall of the sinus, thus following in a general way the course of infection. On several occasions subsequent to a sinus thrombosis operation, the writer has drained a cerebellar abscess through the inner sinus wall.[23]

CASE XX.

J. J.: Perisinus Abscess. Male. Acute Otitis. Six weeks previously symptoms of mastoid involvement. Operated. Perisinus abscess found. Sinus not opened. Did well for eight days, but headache upon moving head. Then irregular vomiting and occasional subnormal temperature. Later, rapid loss of flesh and gradually semi-comatose. Operated on twelfth day. Thrombosis of lateral sinus found and evacuated. Next day deep coma. Without an anesthetic a cerebellar abscess was evacuated through the inner sinus wall. Relief instantaneous. The patient, who had not spoken for two days, and who, during twenty-four hours had been in deep coma, opened his eyes and became entirely rational. Did well for three days, but abscess did not drain well because of small size of opening. Fourth day chill with high temperature, which was repeated. Internal jugular ligated. No clot found. Death ten hours later.

**Comment.**—Although it was not recognized at the time, the thrombus was probably secondary to the abscess, as there were no symptoms of sinus thrombosis. The subsequent chill with high temperature was due to a septic leptomeningitis.

In another case without sinus symptoms, the removal of the bone over the sinus uncovered a localized collection of pus apparently on the sinus; examination revealed the inner surface of the abscess to be the cerebellar cortex, the sinus walls having undergone necrosis.

**Exploration Behind the Sinus Over the Lateral Lobe of the Cerebellum.**—If the abscess is in the lateral lobe of the cerebellum, the dural exploration may be made behind the sinus, uncovering both cerebellar hemispheres.

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[23] Eagleton, W. P.: Brain Abscess of Otitic Origin, with Report of Two Cases, and an Analysis of 131 Cases Reported in Literature; *Transactions of Medical Society of New Jersey*, 1902, p. 194.

Cerebellar abscesses originating from thrombosis of the lateral sinus, while generally located on or in the posterior two-thirds of the cerebellum, are frequently so situated, on either the upper or the under surface of the cerebellum, as not to be accessible by *puncture* from behind the sinus.

**Influence of Displacement of the Affected Hemisphere Beyond the Median Line.**—Failure to appreciate the abnormal position occupied by the affected hemisphere has led me on several occasions to attempt investigation of the cerebellar surfaces after uncovering only the affected hemisphere. Opening the dura was invariably accompanied by herniation of the brain to a sufficient degree to prevent thorough inspection.

*Ventricular Puncture,* by reducing the increased intracranial pressure will prevent destructive herniation, but displacement of the affected lobe beyond the median line necessitates incision of the dura over both lobes, to allow of proper inspection.

**Technic of Exposure behind the Sinus.**—If the cerebellum over the lateral lobes is to be exposed, the opening in the bone must extend horizontally from the posterior edge of one lateral sinus to the opposite sinus; and laterally, from below the superior curved line to the foramen magnum. In removing the bone troublesome hemorrhage from the mastoid emissary and diploic veins may occur. In cerebellar abscess the intracranial pressure is not only increased by the abscess, but also by an internal hydrocephalus—a usual complication of cerebellar abscess.[34] The increased intracranial pressure frequently obliterates the lumen of the large venous sinuses; the blood, ordinarily carried by the sinuses, leaving the skull by the normally small diploic vessels. It is impossible to open the dura without immediate prolapse of the brain and rupture of the piarachnoid. If, however, a ventricular puncture is performed before the dura is opened, the sinuses become patent, the bleeding stops and the cerebellum drops back, permitting dural incision without uncontrollable prolapse of the brain. The dural incision, to admit of inspection, must uncover *both* cerebellar hemispheres, extending from one lateral sinus to that of the opposite side.

**Situation of Abscess Secondary to Sinus Thrombosis.**—No absolutely uniform relation that may be utilized surgically, exists between the point of infection and the situation of the abscess.

Intracerebellar and intrapiarachnoid abscesses are rather uniformly distributed over all surfaces and in all parts of the lateral hemispheres of the cerebellum. The middle lobe, however, is only infrequently the site of an abscess. Metastatic abscesses from pulmonary suppuration occur within the middle lobe, but of the other four recorded cases of middle lobe abscesses none are above suspicion of not being either metastatic in origin, or of having originated from explorative punctures of the cerebellum.

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[34] See examination of the pathological findings in 125 cases of cerebellar abscess, p. 105.

### SITUATION OF ABSCESS IN CEREBELLUM IN 125 AUTOPSY RECORDS.

| | *No. of Cases.* |
|---|---|
| On or near Anterior Surface of Cerebellum | 30 |
| Whole of Lateral Hemisphere | 11 |
| Not Stated | 36 |
| In Posterior Two-thirds of Cerebellum | 23 |
| On or near Lower Surface | 6 |
| On or near Lower and Mesial Surface | 3 |
| On or near Posterior and Mesial Surface | 1 |
| On or near Upper Surface | 1 |
| Lateral Surface | 1 |
| Opposite Lobe | 2 |
| In Middle Lobe | 3 |
| Total | 117 |
| Multiple Abscesses | 10 |
| Bilateral | 2 |

*Surgical Requirements for Systematic Exploration and Evacuation.*—In the treatment of cerebellar abscess secondary to sinus thrombosis, to meet the surgical requirement that the operative procedure may be attended by a systematic exploration which will admit of a uniform operative localization of the suppuration and its complete evacuation, or eradication, it would be necessary in all cases:

(1) *To obliterate* and doubly ligate the descending portion of the lateral sinus.

(2) To expose the dura of the whole cerebellar fossa of the affected side, and as the affected hemisphere occupies a position beyond the median line, the bone over the unaffected hemisphere should be freely removed.

(3) To perform a ventricular puncture in order to relieve the internal hydrocephalus.

(4) Then to incise the dura, as far forward as possible, the incision extending backward through the obliterated sinus and continued in whichever direction is necessary.

Because of the variety of situations of cerebellar abscess, as found at post-mortem, no other surgical manipulation promises uniformly to locate the abscess or to allow of its complete evacuation or eradication.

*Association of Extradural with Cerebellar Abscess.*—At the completion of the intradural operation, the adjacent surface of the petrous pyramid should be inspected. Friedrich[25] and Hegener[26] assert that extradural abscess is the most frequent cause of cerebellar abscess. Although the author's experience does not substantiate this, he must confess to having on two occasions overlooked an associated extradural abscess during operation.

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[25] Friedrich: Die Eiterungen des Ohrlabyrinthes. Monograph. Wiesbaden.
[26] Hegener: Verhandlungen der Deutschen Otologischen Gesellschaft; 1907.

# CHAPTER VII.

# FRONTAL LOBE ABSCESS.

## *Introductory Note.*

**The author's experience with frontal lobe abscess is limited to several cases of adjacent abscess and several of traumatic origin.**

**The number of adjacent frontal lobe abscesses coming under the observation of any one man necessarily must be so limited as to preclude authoritative statement from him. This section, therefore, is based upon a review of the 145 or 150 cases reported in the literature of the subject, with application to their treatment on the lines of the principles expounded in the body of this work.**

**Having been begun with only a few pages recording the personal observations of the writer, this part of the book has expanded to its present proportions after a survey of the literature had revealed repeated errors and hitherto unappreciated viewpoints, and brought to the author's mind a realization of the necessity for an exhaustive study of frontal lobe abscess.**

### ANATOMICAL AND PATHOLOGICAL CONSIDERATIONS PECULIAR TO THE FRONTAL REGION; THEIR SURGICAL IMPORTANCE.

Abscess of the frontal lobe presents surgical problems different from those involved in abscess situated either in the temporo-sphenoidal lobe, or in the cerebellum.

The following anatomical and physiological peculiarities of the frontal bone influence its occurrence and course:

(1) The mechanism of the nose and paranasal sinuses is for the protection, not only of the upper respiratory tract, but of the brain as well. By the aid of its ciliated epithelium, its mucous glands, and its adherent elastic-tissue tunica propria, it adequately prevents the invasion of the bone by repeated adjacent infections within the nose and accessory sinuses.

(2) When the protective mechanism of the nose is overcome, the protective mechanism of the brain tends to limit the infection, first to the dura—pachymeningitis externa—and, failing in this, to the subdural space—pachymeningitis interna. Both protective mechanisms must be overcome before an abscess develops. Thus, in spite of the frequency of nasal suppuration, intracranial complications of nasal origin are very rare.

(3) The compact bony structure of the roof of the orbit, of the

ethmoid, sphenoid, and inner plate of the frontal sinuses, without diploic tissue, contributes to the protection, the compact bony plates offering a bulwark against infection. The absence of cancellous tissue eliminates a harbor for the growth or latency of micro-organisms, such as is furnished by the mastoid. If, however, the infection invades the compact bony plate, extensive nutritional death from necrosis, not caries, results. Necrosis of the compact tissue of the posterior frontal wall of the roof of the orbit or ethmoid is frequently accompanied by the formation of an extradural abscess which, clinical experience teaches, is frequently followed by or associated with an adjacent abscess of the frontal lobe. Anatomically, this association would be expected in this region.

(4) Direct vascular paths run from the mucosa of the accessory sinuses through the bone into the dural plexus, communicating with the veins of the cortex and the longitudinal sinus.

(5) There exists, in addition, an intimate vascular and nutritional relationship between the frontal bone and the dura, covering the frontal lobe.[1] Consequently, infection of the vessels of the diploë of the frontal bone—osteomyelitis—is associated with subperiosteal and extradural abscess, frequently with sinus thrombosis, and occasionally with intradural abscess.

Isolated irritation of the inner layer of the dura stimulates the bony growth of the overlying skull, as shown clinically by the frequency with which dural endothelioma is covered by a bony exostosis. The presence of an exostosis of one of the flat bones of the skull warrants a diagnosis of an adjacent dural endothelioma.

These anatomical conditions render the frontal lobe a unique field, which—when infected—gives rise to pathological changes and clinical manifestations not common in other parts of the skull.

The frontal lobes of the brain also possess anatomical and physiological peculiarities which markedly influence suppuration within the dura, and which render the diagnosis and treatment of frontal lobe abscess especially difficult:

(1) The position and anatomical relationships of the frontal lobes make them especially liable to trauma.

(2) The frontal lobes form the "silent area" of the brain. This largely accounts for their remarkable tolerance of trauma.

(3) The frontal tips are attached to the dura by minute blood vessels, which assist in holding it in position. These are easily rup-

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[1] Skillern, Penn G., Jr.: Rib Cartilage Transplant for Saddle Back Nose; *Annals of Surgery*, 1918, Vol. 68, p. 580—"The frontal lobe receives its nourishment largely from the dura."

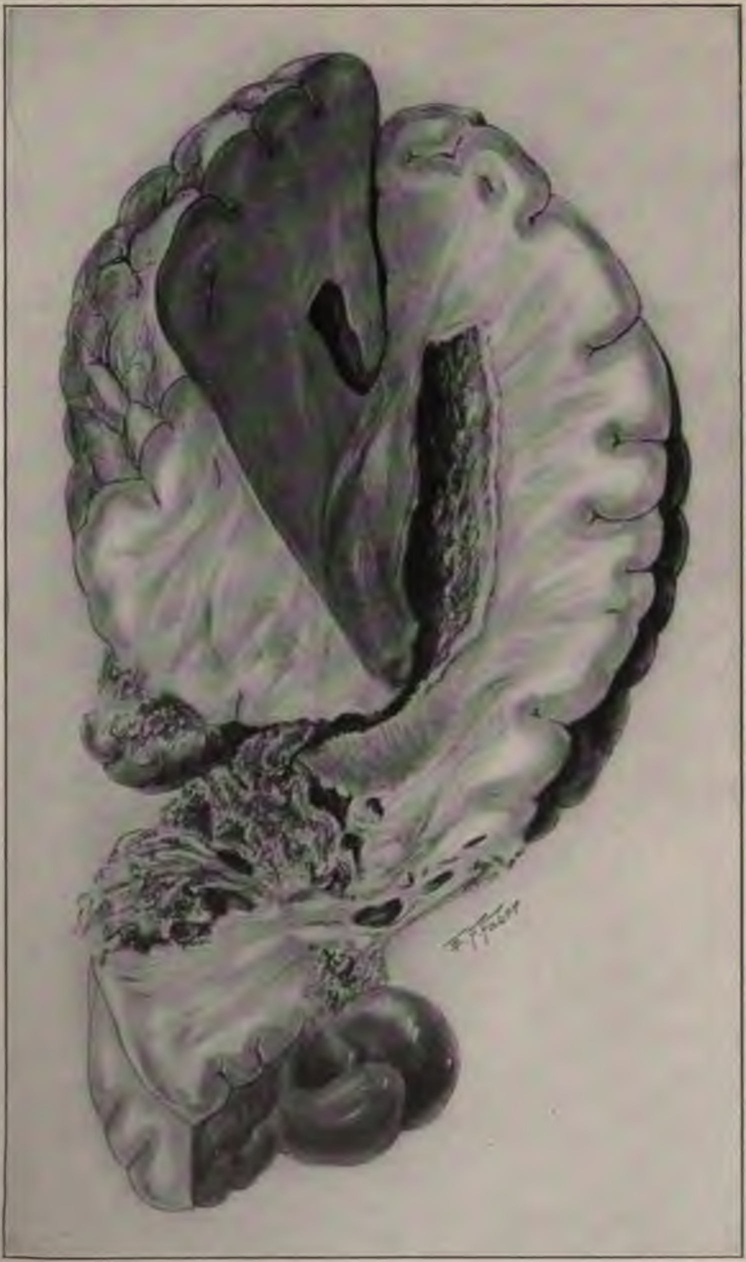

FIG. 29.—Frontal lobe abscess and perforation into lateral ventricle, with
bsequent cerebral hernia. Area of infiltration extending from the hernia into
e ventricle. Lateral ventricle of affected side was filled with pus, which was
mpletely encapsulated within the lateral ventricle—"encapsulated empyema of
e lateral ventricle."

tured by trauma, causing hemorrhage into the pia and subarachnoid, and rendering the frontal lobe especially liable to infection whenever the protective mechanism of the nose and dura is broken through.

(4) The limited area of the frontal lobe admits of the formation of only a small collection of pus without involving the cerebral ganglionic masses, or perforating into the ventricles or the subarachnoid space. Perforation into ventricle or subarachnoid space is rapidly followed by death. In guarding these structures nature generally succeeds in limiting frontal lobe suppuration to a small compass. While a few large ones are recorded, the vast majority of frontal lobe abscesses are of small size and low virulence, being frequently latent, remaining dormant for a considerable period before presenting outstanding symptoms—frequently not until rupture into the ventricle or piarachnoid ushers in the fatal termination. (Fig. 29.)

The protective mechanism of the brain apparently succeeds in attenuating the virulence of many micro-organisms which have entered the brain substance, as a certain proportion of the brain abscesses are sterile on evacuation, and the great majority of frontal lobe abscesses are of low virulence.

(5) The anterior portion of the frontal lobe derives its blood and nourishment from the branches of the anterior cerebral artery, which is, itself, a loop. In the presence of localized suppuration in the frontal region, the reactive forces, having been already subjected to strain in its encapsulation, the development of meningitis, general or localized, is a much more frequent occurrence than during the formation of brain abscess in the temporo-sphenoidal lobe. Therefore, when adjacent frontal lobe abscess has developed, any additional disturbance—extension of the abscess or injury to the cerebral tissue during its evacuation—is badly borne because of the poor blood supply of the frontal lobe:—witness the high mortality following operations for frontal lobe abscess.

### INFLUENCE OF TRAUMA.

Trauma plays a much more important part in the production of frontal lobe abscess than of abscess in any other part of the brain. Given symptoms of increased intracranial pressure, with history of trauma in the frontal region, abscess of the frontal lobe is to be suspected. The explanation for this may be found in the relatively frequent disturbance of the vascular supply of the frontal lobe. Throughout the skull the dura is held firmly to the bone by fibrous connective tissue; the brain, however, is attached to the dura by the blood vessels and nerves. In the frontal region the anterior poles of the brain are attached to the dura by small veins which, after ramifying on the cerebral cortex, become part of the dural plexus. A certain amount of movement of the brain as a whole is allowable between the brain and the dura, the movement being in the subdural and subarachnoid spaces and in the brain itself.

In severe concussion or contrecoup violence, however, the displaced brain's weight is thrown against the frontal poles, causing sufficient displacement to rupture the minute vessels holding it to the dura and causing pial and subarachnoid hemorrhages. With milder forms of injury the anterior frontal poles of the brain are frequently the site of hemorrhage in the pia and subarachnoid space; while a violent injury to the skull is generally productive of a disorganization of the cerebral tissue of the region affected. The writer does not recall a case of a fractured skull which did not show hemorrhage into the pia and piarachnoid space of the frontal poles.

Similar hemorrhage occurs, though much less frequently, in the temporo-sphenoidal tips. On the other hand, the vessels of the dura itself are not injured without direct violence because of its firm attachment to the bone. A rupture of the middle meningeal artery is always associated with a tear in the integrity of the bone.

Trauma of the frontal lobe, however, does not give rise to abscess unless associated with infection, although recently cases have been reported in which transmitted trauma from shell shock with but slight external injury has produced frontal lobe abscess. This is of the greatest surgical importance, demonstrating that transmitted trauma without bodily injury, may give rise to such a degree of cerebral disorganization of the frontal lobe as to cause abscess formation. The lowered resistance permits the micro-organisms from a distance to be deposited by the circulation.[2] While trauma alone is well borne, the frontal lobes are unusually susceptible to a combination of infection and trauma; thus compound fracture of the skull, with involvement of the frontal sinus, is followed by suppurative meningitis in a larger proportion of cases than in other regions.

### RELATIONSHIP BETWEEN OSTEOMYELITIS, EXTRADURAL AND INTRADURAL ABSCESS.

As there exists an intimate nutritional relationship between the diploë and the dura of the frontal bone, and through it with the cerebral tissue itself, any disturbance of the nutrition by infection or

[2] Damaye, Henri: Abscès à staphylocoque du lobe frontal favorisé par la fatigue et le bombardement; *Le Progrès Médical*, 3me. Série; 1918, Tome 33, p. 402.

trauma of the frontal region is apt to be associated with extradural and frontal lobe abscess.

The following pathological associations are of surgical importance: (1) Osteomyelitis of the frontal bone, originating from paranasal suppuration, is comparatively frequent, although it is almost an unknown condition in the other flat bones of the skull. I have seen it attack the temporal bone from aural infection in but one case. It follows extension of infection from the frontal sinus, or more frequently trauma during a frontal sinus operation.

(2) Intradural abscess is associated with extradural abscess in a considerable proportion of cases of infection from the nasal sinuses, from the orbit, or from a complicating osteomyelitis of the frontal bone. In the temporo-sphenoidal region, although an extradural abscess is of frequent occurrence, it is but rarely complicated by a brain abscess.

In the process of limiting the suppurative focus external to the dura, the resisting powers of nature, as a rule, prevent its spread into the cranial cavity. In the frontal region, however, the association of extradural with intradural abscess is so frequent, that it would appear as if an extradural collection either favors or is coincident with the formation of a brain abscess itself.[3]

The uncovering of an extradural abscess during an operation around the mastoid never in itself suggests the advisability of opening the dura; but operative experience has taught the author that, having found an extradural abscess of the frontal region, an intradural abscess is to be suspected. He has therefore come to regard the operative finding of an extradural abscess adjacent to an infected sinus a distinct indication for the exploration of the brain itself.

CASE XXI.

D. McS. Male: Adjacent Frontal Lobe Abscess Following Frontal Sinus Disease, Orbital Phlegmon, or Osteomyelitis, and Associated with Extradural Abscess.

**Previous History.**—Pneumonia five years before; always had some "nose trouble," with slight discharge, since childhood; had lost sense of smell for past three years.

About three weeks before admission to hospital suddenly had pain and swelling of right eye; swelling extended to other eye and then subsided; two weeks later pain in head, with recurrence of swelling of right eye.

**Examination.**—Slight exophthalmos of right eye, with beginning chemosis and diplopia; limitation of motion toward nasal side. Polypoid degeneration of mucous membrane of nose, polyps filling both nostrils; creamy pus in both nostrils, apparently from ethmoids.

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[3] Gerber, P. H.: Die Komplikationen der Stirnhöhlenentzündungen; *Beiträge zur Anatomie, Pathologie, und Klinik der Stirnhöhlen*. Berlin, S. Karger, 1909. (Collected 65 frontal lobe abscesses, 25 or 40 per cent. of which had an associating extradural abscess. He found but 28 cases reported of extradural abscess alone.)

**X-Ray Findings.**—Frontal sinuses moderate size, multilocular, deficiency of illumination both sides, more marked on right; haziness right ethmoid.

**Diagnosis.**—Orbital abscess from ethmoid suppuration.

**Operation.**—Right-sided orbital abscess evacuated; caries of orbital wall of anterior ethmoid cells; ethmoids and frontal sinus filled with pus; pus evacuated.

Examination of pus from sinus revealed large Gram-negative bacilli.

Patient did well for three weeks. Exophthalmos slowly receded. Discharged from hospital.

**Subsequent History.**—Several days later profuse discharge from wound, with severe pain in head, starting from bridge of nose and extending over top to back of head. Temperature and pulse normal; forehead puffy and tender; no tenderness over wound, which was now almost entirely closed.

**X-Ray Findings.**—Frontals, right post-operative, left, marked deficiency of illumination.

**Second Operation.**—Left frontal sinus opened; found to contain pus; thorough removal of mucous membrane, followed by tight closure; right frontal sinus re-opened; large area of osteomyelitis of frontal bone found, osteomyelitis extending into orbit, with necrosis of roof of orbit; small sequestra removed; dura beneath osteomyelitis covered by flattened granulations.

Following second operation patient did well for a few days; suddenly intense headache. Lumbar puncture; smear negative; culture negative—no bacilli. Following day, acute delirium, deep coma and sudden death on second day.

## SURGICAL CLASSIFICATION.

Frontal Lobe Abscesses, like abscess of the cerebellum, should be divided into: I—Chronic Adjacent (Secondary); II—Acute Adjacent (Secondary); III—Intercurrent (Tertiary) to sinus thrombosis or progressive osteomyelitis; IV—Traumatic; V—Metastatic.

### ADJACENT FRONTAL LOBE ABSCESS.

**Occurrence.**—Adjacent frontal lobe abscess is of very infrequent occurrence, there being not more than 150 cases reported in the literature of the subject, while there are records of over 900 cases of adjacent abscesses of the middle fossa.

It will be asked why it is that, with nasal suppuration almost universal and aural suppuration comparatively infrequent, the number of adjacent brain abscesses of otitic origin are many times more numerous than those of nasal origin.

The explanation is simple—*the nose and accessory sinuses are essentially a mechanism for the protection, not only of the upper respiratory tract, but of the underlying bone of the nose and paranasal sinuses.* On the other hand, the mucous membrane of the antrum and the mastoid cells is merely a cellular covering of the bone, which is easily destroyed, thereby allowing invasion of the bone.

Histologically the lining structure of the bony cavities of the nose and paranasal sinuses is made up of mucous glands and vascular erectile tissue containing lymphatic tissue or vessels covered by ciliated columnar epithelium, with a connective and elastic tissue stroma firmly adherent to the underlying periosteum. Each of these elements is designed for the prevention or removal of infection,—the secretion from the glands for encapsulation and attenuation of micro-organisms and foreign substance, the lymph tissue for their assimilation, the erectile tissue for the mechanical obstruction to their entrance, and the cilia for their removal. Even after infection of this lining structure itself, the hypoplastic mucoid degeneration, so-called, carries on the process of protection, while the elastic lamina propria, with its firm adhesion to the bone, prevents the micro-organisms from attacking the bone.

Other factors must be added to the usual infective diseases of the nasal sinuses before the brain can be involved from the sinuses. These factors are trauma, from blow or fall, or coincident with operation on the sinuses; infection by virulent micro-organisms; and acute exacerbation of a chronic process by re-infection, or the assuming of virulence by attenuated micro-organisms, when they reach or are transplanted on previously unaffected tissue, such as compact tissue of the bone, the dura, or the piarachnoid.

Trauma and renewed virulence generally act together, as during operation or in fracture of the inner plate.

Virulent infection, or greatly lowered resistance such as follows scarlet fever or diphtheria, may cause a complete disorganization of the soft tissue lining the sinuses, with perforation of the outer wall of the ethmoid cells or of the anterior wall of the frontal sinus, associated with bony necrosis, with or without extension into the cranium.

In a large number of reported cases frontal lobe abscess was preceded by an acute suppuration of sufficient intensity to cause redness, or a bony necrosis of the outer frontal sinus wall and the lamina propria of the ethmoid, with an associated orbital phlegmon.

If, during operation, the frontal sinus is forcibly curetted, thereby not only removing the chief barrier, but liberating micro-organisms which have been attenuated by encapsulation in the mucous secretion and tissue, and transplanting them into the vascular spaces and lacunæ of healthy bone, whereby they assume virulence, there may result osteomyelitis, perivasculitis, or retrograde thrombophlebitis, eventuating in pachymeningitis interna, suppurative leptomeningitis, sinus thrombosis, or brain abscess.

Milligan,[4] in forty frontal sinus operations, encountered frontal lobe abscess in two, or five per cent.

In abscess following acute virulent infection, the intense pain of bony involvement is of diagnostic value, as it will precede the clinical manifestations of cerebral extension. When the abscess follows the trauma of an operation, the pathological process being perivascular, there may be no complaint of pain.

**Origin of Infection.**—In adjacent frontal lobe abscess the original site of infection is from the nasal accessory sinuses or the orbit, or from a complicating osteomyelitis of the frontal bone itself, originating from either the sinuses or the orbit.

Of the adjacent frontal lobe abscesses originating from the nasal sinuses, the vast majority follow suppuration of the frontal, or of the frontal and ethmoids together; a few are reported as originating from the ethmoids and sphenoid, but none from infection of the sphenoidal sinuses alone.

As the ethmoid mucous membrane is separated from the dura only by a thin layer of compact bony tissue, suppuration of the ethmoids would naturally be regarded as a frequent cause of brain abscess or meningitis; in reality the ethmoids alone but infrequently cause intracranial complications, despite the fact that the ethmoids are the frequent site of operative procedure. This immunity is maintained because the nutritional supply of the superior surface of the ethmoid bone itself is not easily disturbed, necrosis of the ethmoid roof being very rare. The operative procedure must be of a gentle character, since perforation of the ethmoid plate is attended with a high mortality. In removal of the mucous membrane it is to be remembered that the attachment of the lamina propria to the bone is loose, the lining membrane being easily removed, while the absence of diploic tissue prevents latent infection.

Ethmoid suppuration, however, by necrosis of the lamina propria, is frequently the cause of an orbital phlegmon. An orbital abscess is the direct cause of frontal lobe abscess in a large proportion of cases —about 12 per cent.

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[4] Milligan, Sir William: Some Practical Points in the Treatment, Operative and Non-operative, of Frontal Sinus Suppuration, with an Analysis of the Ultimate Results Obtained in Forty Cases; *Journal Laryngology*, August, 1904, Vol. 19, p. 439.

Milligan, Sir William: Suppurative Frontal Sinusitis; *British Medical Journal*, 1905, I, p. 171.

### PATHS OF INFECTION.

In adjacent abscess, infection extends either by (1)—direct continuity of tissue suppuration; (2)—through thrombosis of veins in the mucosa (thrombophlebitis); (3)—through a combination of continuity of tissue suppuration and thrombophlebitis; or, (4)—through a complicating osteomyelitis.

(1)—*Infection by Continuity of Tissue Suppuration:* (*a*) Examination of the reported cases of frontal lobe abscess shows that in numerous instances the diseased mucous membrane of the frontal sinus was in direct contact with the brain itself through necrosis of the dura and of the posterior sinus walls (an intracerebral abscess with a stalk).

With an intact dura a piarachnoid abscess may be formed, adjacent to a necrotic posterior wall; the intrapiarachnoid abscess being the result of an infection of the cerebro-spinal fluid, called by the toxic irritants in the infected area to protect the brain.

On account of the anatomical peculiarities of the nasal sinuses, while the mucous membrane is frequently infected, the bone itself is comparatively rarely involved; when attacked, however, it is the seat of necrosis from nutritional death—not caries—as the structures are made of one firm layer of compact bony tissue, and not, as in the temporal bone, of cancellous tissue with compact tissue boundaries. In consequence of this absence of soft, medulla-filled cancellous tissue, the frontal bone does not furnish a harbor for bacterial growth as does the temporal bone, the structure of which favors caries and latent infection. For the same reason ebonization, a reparative, but pathological process of the mastoid, is not encountered in the nasal accessory sinuses.

The mucous membrane of the frontal sinus is more firmly held to the periosteum of the bone by the tunica propria than is that of the ethmoid, while the mucous membrane of the sphenoid is comparatively loosely attached. Therefore bacterial invasion into the tunica propria itself is much more apt to be followed by necrosis in the frontal than in either of the other sinuses, although the free venous drainage of the sphenoidal mucous membrane into the adjacent cavernous tissue is its chief protection.

(*b*) Onodi describes "half ethmoidal canals," through which the ethmoidal vessels pass and in which the mucous membrane of the nose comes in direct contact with the dura and with the periosteum of the orbit, thus favoring extension by continuity.[5]

[5] Onodi, A.: Ueber die okulo-orbital intrakraniellen und cerebralen Komplikationen nasalen Ursprungs; *Medizinische Klinik*, 1914, No. 17, p. 719.

(*c*) Dehiscence in the bony structure of the cells themselves likewise favors extension of infection by this path.

(2)—*Infection by Retrograde Thrombophlebitis or Perivasculitis* of the minute veins in the mucosa of the frontal sinus which communicate freely with the dural and pial veins and with the venous sinuses. This, i. e., thrombophlebitis, is the path of infection in a large proportion of cases. It is associated with an intact posterior wall of the frontal sinus, and within the dura the abscess is separated from the overlying dural membrane by apparently normal cerebral tissue; or it occupies the subdural space.

Although a certain proportion of the recorded cases give the lymph vessels as the path of infection, this claim is not substantiated by microscopical evidence.[6, 7]

(3)—*Infection by a Combination of Tissue Suppuration and Thrombophlebitis:* In this case the tissue suppuration causes an involvement of the posterior wall of the frontal sinus, while the thrombophlebitis is the originating cause of the intracerebral abscess.

(4)—*Infection by a complicating Osteomyelitis,* when the frontal lobe abscess may be either an adjacent paranasal sinus brain abscess, or, what is more frequent, secondary to osteomyelitis. In the latter case the abscess may be far removed from the nasal sinuses. Osteomyelitis of the frontal bone, following acute infection or operation upon the frontal sinuses, is primarily a thrombotic process involving the soft vascular layers of the diploë. In its extension both the inner and outer tables are involved. Consequently osteomyelitis is always associated with a sub-periosteal and extradural abscess[8] and with necrosis of the compact bone of the roof of the orbit. The involvement of the veins themselves is generally far beyond the apparent area of bony involvement. Failure of complete eradication of the process during operation on the skull is largely responsible for its subsequent recurrence.

[6] Turner, Logan A.: The Lymphatic Apparatus of the Nose and Nasopharynx in Its Relation to the Rest of the Body; *Verhandlungen des 3ten. Internationalen Laryngo-Rhinologischen Kongresses,* Berlin, 1911, S. 65. (Reviews literature and finds no positive evidence of invasion of the brain by lymphatics.)

[7] Zwillinger, Hugó: Experimentelle Untersuchungen zur Mechanik der intrakraniellen und cerebralen Komplikationen der Stirnhöhlenentzündungen; *Arch. für Laryngologie und Rhinologie,* 1913-14, Bd. 28, S. 271. (Zwillinger, by experimental injection of the subdural space, demonstrated certain irregular vascular channels running from it into the mucosa of the frontal sinus and ending in irregular buds in the mucous membrane. These he regarded as lymphatic channels. Experimentally he failed to demonstrate a lymphatic plexus in the frontal sinus, or to show whether the channels ran through the separate openings in the bone or not. An examination of his paper and its accompanying drawings is far from convincing that the injected channels were lymphatic.)

[8] Schilling, R.: Ueber die Osteomyelitis des flachen Schadelknochens und des Mittelohres; *Zeitschrift für Ohrenheilkunde,* 1904, Bd. 48, S. 52. Ergänzungsheft.

### ADJACENT FRONTAL LOBE ABSCESS ASSOCIATED WITH OSTEOMYELITIS.

The dura offers firm resistance to infection of the intracranial contents; but my experience teaches that when osteomyelitis leads to necrosis of the bone of the orbit or the posterior sinus wall, because of the intimate nutritional and vascular supply of this region the development of extradural abscess in this area is apt to be associated with adjacent intradural suppuration.

*Intercurrent (Tertiary) Brain Abscess, Associated with Parasinoidal Suppuration but Secondary to Osteomyelitis, Longitudinal, or Cavernous Sinus Thrombosis, and Situated at a Distance from the Primary Site of Infection:* In osteomyelitis from nasal infection a thrombophlebitis of the diploic and dural vessels occurs at a considerable distance from the area primarily infected. This thrombophlebitis may extend inside the dura, causing either a brain abscess or, more frequently, longitudinal or sinus thrombosis, which in turn may be the immediate origin of a localized intradural collection of pus. The immediate origin of the brain abscess determines its location within the cerebral substance, just as thrombosis of the lateral sinus, when the direct cause of cerebellar abscess, determines the site of the abscess, placing it on any surface or on any part of its tributaries, possibly remote from the original site of the primary infection, on the petrous bone.

In the determination, then, of the location of a brain abscess, the immediate originating pathological process—osteomyelitis, longitudinal or cavernous sinus thrombosis—should be considered as if it were secondary to a complicating condition. The abscess may be situated at a great distance from the nasal sinuses. In a case reported by Knapp a brain abscess secondary to a longitudinal sinus thrombosis was situated near the torcular, although the primary infection was in the accessory sinuses.[9]

### *Types of Frontal Lobe Abscesses.*

I—Chronic Adjacent (Secondary);
    (a) Intracerebral without macroscopical evidence of cortical involvement;
    (b) Intracerebral with a stalk;
    (c) Meningeal.
II—Acute Adjacent (Secondary);
    (a) Intracerebral without macroscopical evidence of cortical involvement;
    (b) Meningeal.

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[9] Knapp, Arnold: Osteomyelitis of the Skull with Empyema of the Nasal Accessory Cavities; Sinus Thrombosis; Pyemia; Death; Autopsy; *Archives of Otology*, 1903, Vol. 32, p. 181.

**III—Intercurrent (Tertiary) to progressive osteomyelitis or longitudinal or cavernous sinus thrombosis;**
  **(1) Chronic;**
  **(2) Acute.**
**IV—Metastatic.**
**V—Traumatic.**

### ADJACENT MENINGEAL, SUBDURAL.

There is clinical evidence that subdural adjacent abscesses are more common in the frontal region than in any other portion of the brain. (See Case VIII—A. M. M., p. 37.) The subdural space apparently possesses great resistance to infection, subdural abscess being infrequent. In frontal sinus disease, however, there is microscopical evidence (Streit)[10] that once the mucous membrane barrier is overcome and the vessels of the bone invaded pachymeningitis interna and small subdural collections of pus are frequently present.

Pachymeningitis interna and small subdural abscesses are nature's effort to limit the process. Either process may not give rise to clinical symptoms sufficient to excite suspicion of its existence; when present, however, any trauma, such as in an operation on the frontal sinus, may occasion a virulent leptomeningitis, an unexpected, frequently terminal development.

von Hinsberg[11] examined microscopically a case in which death from meningitis rapidly followed an operation for frontal sinus disease. He found that without microscopic evidence of necrosis the bone was traversed by numerous small vessels, some of which were thrombosed, the process evidently being of long standing. Between the dural layers were several large cavities filled with albuminous fluid and microörganisms. Two of these cavities communicated with the subarachnoid space, a direct connection between the outer sinus wall by way of the thrombosed vessels being demonstrated. One focus remained encapsulated against the subdural space while others perforated it, occluding the subdural and the subarachnoid spaces and thus preventing infection. At the operation these were broken down, causing a purulent leptomeningitis.

Streit[12] demonstrated experimentally that a subdural abscess limited to the subdural space can be produced under favorable conditions, the subdural space itself being generally skipped in the extensive infective process from the frontal sinus to the subarachnoid. He presumes that a subdural abscess probably is the second stage in the development of leptomeningitis. The value of Streit's and von Hinsberg's microscopical and experimental work lies in its demonstration that pachymeningitis interna and small foci of subdural pus, which have not given rise to symptoms and whose presence is consequently unsuspected, may exist, and, by the trauma of operation on an infected frontal sinus, may give rise to virulent leptomeningitis or brain abscess.

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[10] Streit, Herman: Weitere Beiträge zur Histologie und Pathologie der Meningitis und Sinusthrombose; *Archiv für Ohrenheilkunde*, 1912, Bd. 89, S. 177.

[11] von Hinsberg, V.: Ueber den Infektionsmechanismus bei Meningitis nach Stirnhöhleneiterung; *Verhandlungen der Deutschen Otologischen Gesellschaft*, 1901, Bd. 10, S. 191.

[12] Streit, Herman: Weitere Beiträge zur Histologie und Pathologie der Meningitis und Sinusthrombose; *Archiv für Ohrenheilkunde*, 1912, Bd. 89, S. 177.

### ADJACENT PIARACHNOID ABSCESS.

The reported cases make it appear that this type of meningeal abscess is not as frequent in the frontal as in the temporo-sphenoidal region, nor does it attain as large a size. These characteristics are probably accounted for by the absence of cancellous tissue in the nasal region, an irritating area of bony caries, consequently, being much less frequent in this region than in the temporal.

Piarachnoid abscess is originally of toxic origin from the irritation of an adjacent bony inflammation—serous meningitis—which later becomes infected by the invasion of micro-organisms into the protective collection of cerebro-spinal fluid.

As in the temporo-sphenoidal region, if the irritating cause is removed before the infection occurs, the collection of cerebro-spinal fluid will disappear. Cases are recorded of the disappearance of the cerebral symptoms by sinus operation alone, without the opening of the dura.

## ADJACENT ABSCESS "WITH STALK."

This type of abscess is of relatively more frequent occurrence in the frontal lobe than in either the middle or the posterior fossa. As with temporo-sphenoidal abscess, and for the same reason, abscess with a stalk offers the best prospect of recovery. Although only twenty cases of frontal lobe abscess have recovered, three have been *spontaneously* cured by rupture and evacuation through a stalk. The establishment or utilization of a stalk—nature's method of cure—in frontal lobe abscess, as in temporo-sphenoidal abscess, should be the fundamental surgical aim. With a stalk present the operative mortality should be low.

## ADJACENT INTRACEREBRAL ABSCESS WITHOUT MACROSCOPICAL EVIDENCE OF CORTICAL INVOLVEMENT.

This type of abscess is generally situated in the white medullary substance, about one-half inch from the cortex. It is usually of a small size and of very low virulence, remaining latent for long periods of time. The literature records a few cases of the abscess growing backwards, involving the speech area on the left side, and then the face and arms.

*Metastatic Abscess* is relatively more frequently encountered in the frontal lobe than in other portions of the brain, and the left frontal lobe is attacked more frequently than the right.[13]

The special predilection of metastatic foci to find lodgment in the frontal lobes would favor the view that the return circulation through the veins of the frontal lobe furnishes a favorable condition for thrombotic-embolic formation—slowness of current and ebbing with poor resistance.[14] This latter condition may be assisted by the anatomical peculiarity of the arterial supply of the frontal lobe, coming as it does from a loop of a loop—the anterior cerebral artery; and therefore the arterial supply, while ordinarily ample, is easily disturbed by traumatic or inflammatory influence.

Among metastatic abscesses should be included all frontal lobe abscesses of otitic origin, as a careful examination of the literature—Lombard,[15] Berens,[16] Schwabach,[17] Nonne,[18] von Hinsberg [19]—will demonstrate that they all possess the diagnostic peculiarities of metastatic abscess, namely, an apoplectiform onset, which although often so mild as to escape notice, still can be elicited by careful examination and location of the abscess at a distance from the primary infection, to which site it has been conveyed by an intercurrent osteo-

[13] Couteaud: Metastatic Abscess in the Brain; *Revue de Chirurgie*, Paris, 1913, Vol. XXXIII, No. 7, pp. 1-172. (States that "The left side and the frontal lobe are most frequently involved.")

[14] Browning, in an examination of 200 cases of supracerebral veins, found 59 in the frontal region, jumping over from the piarachnoid to the dura at 3-4 cm. from the longitudinal sinus, and only 9 in the posterior, superior and central; thus, he thinks, favoring infection. Browning, W.: The Veins of the Brain and its Envelopes: their Anatomy and Bearing on the Intracranial Insulation; Monograph, Brooklyn, 1884.

[15] Lombard, Bloch A., and Moulouguet, A.: Un cas d'abscès frontal du côté opposé à une otite suppurée chronique (Frontal Lobe Abscess of Otitic Origin); *Annales des Malades de l'Oreille, etc.*, 1914, Tome 90, p. 749.

[16] Berens, T. Passmore: Abscess of Frontal Lobe of Otitic Origin; *Annals of Otology, Rhinology and Laryngology*, June, 1913, Vol. XXII, p. 433.

[17] Schwabach, D.: Beitrag zur pathologischen Anatomie des inneren Ohres, und zur Frage vom primären Hirnabszess; *Beiträge zur Ohrenheilkunde*, Lucae Festschrift, 1905, S. 55.

[18] Nonne, M.: Ueber Fälle von benignen Hirnhauttumoren; ueber atypisch verlaufene Fälle von Hirnabszess, sowie weitere klinische und anatomische Beiträge zur Frage vom "Pseudo-tumor Cerebri"; *Deutsche Zeitschrift für Nervenheilkunde*, 1907, Bd. 33, S. 316-357.

[19] von Hinsberg, V.: Ueber den Infections-Mechanismus bei Meningitis und Stirnhöhleneiterung; *Verhandlungen der Deutschen Otologischen Gesellschaft*, 1901, S. 191. (Gives microscopical findings of path of invasion by blood vessels and lodgment in subdural space, with microscopical illustrations.)

myelitis or the involvement of a large venous channel, such as the longitudinal sinus.

The surgical importance of the relative frequency of metastatic abscess being situated within the frontal lobe, especially on the right side, should be more generally appreciated, as in a case in which a metastatic abscess is suspected (with symptoms of cerebral suppuration but without localization), the operative exposure should be large enough to admit of exploration of the frontal lobes, especially of the left.

## TRAUMATIC FRONTAL LOBE ABSCESS.

As would be expected from its exposed position, traumatic abscess is relatively common in the frontal lobe, and for the reason that this area is silent, the encapsulated suppuration is apt to be overlooked until a fatal meningitis has developed. (See Case XXII, p. 125.) Experience has taught the author that, given a history of trauma of the frontal region, especially if a fracture of the frontal bone is known to have occurred, slight but continued symptoms—such as headache, slight changes in disposition, dizziness, etc.—call for an exploration of the frontal region. In the author's experience the results of operative treatment of localized frontal lobe abscess of traumatic origin are most satisfactory, the operative mortality being very low, in contradistinction to the high recorded mortality in adjacent frontal lobe abscess.

### IMMEDIATE OCCASION OF ADJACENT ABSCESS.

In a large proportion of the reported cases the adjacent frontal abscess has been preceded by operative trauma on an accessory sinus. So uniformly has this occurred that it is necessary to assume either that the operation on the sinus was the immediate occasion of the intradural suppuration, or that the intracranial origin of the symptoms was not appreciated by the surgeon at the time of the sinus operation.[20]

While the latter supposition indubitably explains many of the cases, the writer is of the opinion that undue trauma during a sinus operation is also frequently responsible. The forcible curetting of the firmly adherent membrane from the posterior wall of the frontal sinus produces a condition similar to an acute virulent infection by liberating into the damaged bony tissue micro-organisms which have been attenuated by the protective mechanism of the accessory sinuses; these organisms again become virulent, attack the blood vessels of the denuded bones and produce a thrombophlebitis with extension into the central tissue by their transplantation into new soil, finding here a favorable medium from the damage done to the tissues by the operator.

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[20] Leegard, Frithjof: Cerebral Abscess of the Frontal Lobe; *Annals of Otology, Rhinology and Laryngology,* March, 1919, p. 115. ("Often the mode of procedure is to operate for the sinusitis. If the symptoms do not disappear, as had been expected, if they continue and increase, it gradually becomes clear that there exists something more than an inflammation in the frontal sinus, and then finally the cerebral abscess is diagnosed. Such a state of affairs is so typical that on reading through the history of cases we find these conditions recurring again and again, almost with the regularity of a law.")

### Situation of Adjacent Frontal Lobe Abscess.

The situation depends upon the point of infection. As the frontal sinus has great variations in its size, the abscess may be situated in any part of the frontal lobe. It is consequently of great importance that a roentgenogram be taken, depicting at least the limitations of the frontal sinus. Among the recorded cases such a high proportion present necrosis of the posterior wall adjacent to which the abscess was situated, that the writer is led to believe that such an inflammatory defect is of greater surgical assistance than is the operative finding of a defect of the posterior. Either in itself is more or less a guide to the location of the abscess. Where the abscess is due to complicating osteomyelitis, longitudinal, lateral or cavernous sinus thrombosis, it may be situated at a great distance from the infecting sinus.

**Presence of Capsule**: Adjacent frontal lobe abscesses follow the same rule as regards capsule formation as do abscesses of the middle fossa; although, on account of the low order of the virulence, the limiting membrane is frequently slight in spite of the long duration of the abscess.

### Operative Result in Recorded Cases of Adjacent Abscess.

An exceptionally high mortality—disproportionately much greater than abscess in any other region of the brain—attends frontal lobe abscess.

Of the 87 cases collected by Boenninghaus,[21] only 15, or 17 per cent. recovered, a mortality of 83 per cent. Of the 15 that recovered three are reported as having been evacuated spontaneously, leaving but twelve out of the 87, or about 14 per cent. of the total number as successfully operated upon. Boenninghaus's compilation thus demonstrates that the surgical means adopted had been inadequate, or had been misdirected, especially as nature, unaided, successfully evacuated 20 per cent. of the total recoveries; whereby the inference is forced that with proper technic the mortality should not be high.

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[21] Boenninghaus: Chirurgie der Nebenhöhlen inkl. Chirurgie der Endokraniellen Komplikationen; *Handbuch der spez. Chirurgie des Ohres und der oberen Luftwege*, Bd. III, Part II, S. 239.

**Treatment.**—As in a large number of the reported cases of frontal lobe abscess the patient died suddenly before a positive diagnosis had been made, operation must be performed while the diagnosis is still largely problematic, and consequently must be more or less exploratory.

*Principles of Exploratory Intradural Operation.*—Surgical procedure must meet two requirements of all intradural explorative operations. It must be through a clean field, so that, with failure to locate the abscess, the dura may be tightly closed; it must be so conducted that direct or indirect damage to cerebral tissue is most improbable.

*Operative Intradural Policy.*—In proposing a policy of surgical procedure for frontal lobe abscess the surgical observations previously described should receive consideration. As the operative results in adjacent frontal lobe abscess have been much poorer than in adjacent abscess of the middle or posterior fossa, while post-operative papillœdema and hernia cerebri are frequent; and as traumatic secondary compression is frequently the cause of the fatal result; and, moreover, while nature, unaided, has cured one-fifth of all the cases that have recovered by spontaneous evacuation—for few deaths result after spontaneous evacuation—therefore, any operative procedure should be planned to evacuate the abscess as nearly as possible after the method of nature.

*Technic of Operations for Suppurative Lesions of the Accessory Nasal Sinuses.*—All operations on the paranasal sinuses should consist of simple drainage through the nose, or of an external operation with delicate but thorough eradication of the diseased mucous membrane, simple drainage being insufficient and forcible curetting dangerous. Streit,[22] von Hinsberg,[23] and Zemann [24] have demonstrated microscopically that pachymeningitis interna and small subdural foci may be converted into a general septic meningitis.

**Termination.**—Cases of adjacent frontal lobe abscess rarely develop compressive symptoms—coma, slow pulse, papillœdema—death generally occurring suddenly while the patient is still active. The absence of compression symptoms until but a few hours or even minutes before death accounts for the large number of unrecognized frontal lobe abscesses, the patients dying without intradural exploration. The immediate cause of death is either unknown or is traceable to rupture of the abscess through the piarachnoid or into the lateral ventricle. With rupture into the piarachnoid space death may be almost instantaneous, convulsions ushering in the demise; or it may be preceded by a virulent meningitis, lasting but a few hours. (See Case, p. 125.) (Fig. 30.)

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[22] Streit, Herman: Weitere Beiträge zur Histologie und Pathologie der Meningitis und Sinusthrombose; *Archiv. für Ohrenheilkunde,* 1912, Bd. 89, S. 177.

[23] von Hinsberg: Ueber den Infektions-Mechanismus bei Meningitis nach Sternhöhleneiterung; *Verhandlung der Deutschen Otologischen Gesellschaft,* Bd. 10, S. 191.

[24] Zemann, W.: Beitrag zur Kenntniss der endokraniellen Komplikationen nach chronischer Nebenhöhleneiterung; *Zeitschrift für Laryngol., Rhinol., und ihre Grenzgebiete,* 1913-1914, Bd. VI, S. 545.

Sudden death frequently occurs without rupture into the ventricle or the subarachnoid space. For some reason, brain abscess in any part of the brain frequently terminates suddenly, the post-mortem failing to reveal either a leak into the ventricles or any other apparent cause for the sudden termination. It is probably due to an acute toxæmia which paralyzes the vital centers, as death is preceded by a sudden elevation of temperature, with respiratory failure.

According to Gerber's [25] collection of recorded cases of frontal lobe abscess, 65 in number, eight recovered. Of the remaining 57, in 21 cases, or 37 per cent., the cause of death was unknown. Of the remainder, with known cause of death, 36 in number, the great majority ruptured into the subarachnoid space. In 13 per cent. of the total number the lateral ventricle was involved. Of the fatal cases in which the cause of death was stated, 88.8 per cent. died of purulent leptomeningitis.

In Möller's case a latent abscess caused death in five hours.[26]

In Pfingst's case, without symptoms of brain abscess, excepting head pains on the side of an orbital suppuration, the patient died suddenly in convulsion. Autopsy showed a large encapsulated abscess, frontal lobe, entirely within the cortical surface.[27]

*Surgical Aims of Operation for Adjacent Abscess of Frontal Lobe.*—The osseous flap should be large enough to permit of inspection of the intradural surface over the posterior wall of the frontal sinus, the roof of the ethmoid, and the orbit, as adhesions between the dura and the brain call for evacuation through the protected area.[28, 29]

The intracerebral manipulation should be confined to, (a) the

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[25] Gerber, P. H.: Die Komplikationen der Stirnhöhlenentzündungen; Monograph, Berlin, S. Karger, 1909.

[26] Schausboe: Ein Fall von Empyema Sinus Frontalis mit Stirnlappen-Abszess; *Trans. Danish Otol. Laryngol. Society,* March, 1915; *Inter. Centralblatt für Laryngol.,* 1915, Bd. XXXI, No. 12, S. 396.

[27] Pfingst, A. O.: Brain Abscess (circumscribed purulent encephalitis), Its Course and Pathology; *Miss. Valley Med. Jour.,* Louisville, 1918, Vol. 25, p. 193.

[28] Elschnig: Der orbitogene Hirnabszess; *Prager med. Wochenschrift,* 1914, Bd. XXXIX, S. 37.

[29] Piffl: Zur Kasuistik der rhinogenen Stirnabszesse; *Prager med. Wochenschrift,* 1914, Bd. XXXIX, S. 39.

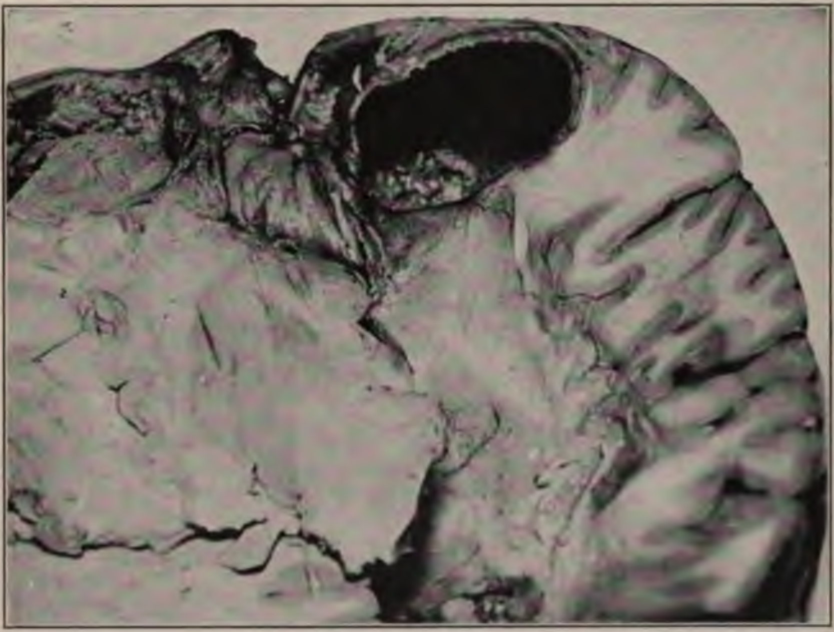

FIG. 30.—Chronic encapsulated intracerebral frontal lobe abscess with stalk, unrecognized although present for months. Sudden death from acute leptomeningitis. No rupture into ventricle. The cause of sudden death in cases of brain abscess without rupture into the ventricle has not as yet been fully explained.

**thorough evacuation of the abscess; (b) the utilization or the establishment of a tract; (c) the accomplishment of both without trauma or infection of the normal cerebral tissue; and (d) the avoidance of secondary compression.**

**Technic.**—The osteoplastic flap should be as large as possible and low down over the frontal avoiding, if possible, the diseased frontal sinus; its base should be external. Ventricular puncture before opening the dura will allow the dura to be incised without prolapse. After incision of the dura the placing of the head over the edge of the table will permit the brain to fall upward and toward the opposite side sufficiently to allow of elevation and inspection of the under surface of the frontal lobe. If adhesions exist between the brain and the dura, the abscess should now be drained through the area of protective adhesions, into the frontal sinus, as recommended in detail in the discussion of temporo-sphenoidal lobe abscess.

In intracerebral abscess without macroscopical evidence of cortical involvement the abscess should be thoroughly evacuated and a tract established, as recommended in the location of temporo-sphenoidal lobe abscess. No attempt at eradication should be made, as the frontal lobe does not stand trauma well on account of the poor circulation.

At the completion of the operation, if the slightest doubt exists that trauma has been occasioned, a large dural opening should be left by the sacrifice of the bony flap, the brain being covered by fascia lata.

*Puncture of Dura and Evacuation of Abscess through Posterior Wall of the Frontal Sinus*—

This is advisable only when an extradural abscess or a defect in the posterior wall is accidentally found at an operation for frontal sinus disease. It is apt to be associated with cerebral trauma, does not admit of complete evacuation of either subdural or intracerebral abscess, or of decompression for secondary œdema.

CASE XXII.

Chronic Encapsulated Frontal Lobe Intracerebral Abscess with Stalk, following Trauma and Secondary Infection of Frontal Sinus.

**Chief Complaint.**—Headache, dizzy spells and defective vision of right eye.

**History.**—Had skull fractured from explosion of stone three months previously. Speaks but little English.

**Examination.**—Old wounds across forehead: Eyes: right cornea, dense scar across centre; Vision 10/70; left cornea, media and fundus normal; Vision 20/20. Ears: hearing,—right, whisper, 20 feet; left, whisper 10 feet. Bone conduction apparently shortened. Rotation to right; past-pointing,—right arm, no past-pointing; left arm, past-pointing to left. Rotation to left; past-pointing,—right arm, past-pointing to right; left arm, no past-pointing. Rotation to right; nystagmus 24 seconds, falling normal. Rotation to left, nystagmus 20 seconds, falling normal. Examination unsatisfactory.

Three months later re-admitted to hospital.

**Chief Complaint.**—Hiccough. States that "nothing bothers him." Headaches; no dizziness. Has been unable to work well since accident.

**Examination.**—Patient speaks but little English, is very unintelligent, and at times appears drowsy. Hiccough at irregular intervals. Temperature and pulse normal. Reflexes normal; no paralysis or anesthesia; no aphasia. Eyes:

Papilloedema bilateral; edges of discs indistinct; veins tortuous. No hemorrhages.

**X-Ray Examination.**—"Old fracture of frontal bone."

Following day hiccough disappeared. Following day vomited, seemed brighter. Suddenly chill followed by convulsions. Temperature suddenly elevated from normal to 105. In deep coma. Lumbar puncture. Fluid turbid. Cell count 19,000. Streptococci. Death within a few hours.

**Post-Mortem Examination.**—Leptomeningitis; abscess of brain of right frontal lobe, with very thin capsule surrounded by several areas of small secondary pus formation. Both frontal lobes degenerated; tip right frontal lobe forced into defect in posterior wall of right frontal sinus, result of old fracture. Mucous membrane of frontal sinus thickened.

**Remarks.**—From the post-mortem examination it would appear that the fracture extended through the posterior wall of the frontal sinus, and was associated with injury to the frontal lobe and herniation of the brain into the frontal sinus. An infection of the mucous membrane of the sinus being now in direct contact with the brain, resulted in the formation of the brain abscess and the development of a purulent leptomeningitis.

## CHAPTER VIII.

## HERNIA CEREBRI.

In the treatment of all intradural lesions the introduction of any drainage material is dangerous. Cushing's painstaking tight closure, in layers, of the operative wound has contributed largely to his low mortality following operation for brain tumor. He has long taught that "backing out," being a reconstructive process, must of necessity consume at least as much time as the destructive approach. In non-suppurating intradural lesions failure of water-tight closure frequently results in hernia cerebri of the fungating variety, which terminates in death from meningitis.

Since it is but rarely possible in operating for brain abscess completely to eradicate the suppuration, some form of drainage is necessary. In a considerable proportion of cases drainage of whatever kind is followed by hernia cerebri. This is particularly apt to occur if the evacuation is not complete,[1] if the trauma of the operation excites severe reactive œdema, or if the dural opening is improperly placed.

The problems involved in the causation, pathology and treatment of hernia cerebri have recently attracted renewed attention because of the frequency of hernia from shell injuries during the world war.

### ETIOLOGY.

In the presence of a dural defect hernia cerebri will follow any increase of the brain's bulk, or increase in the intracranial but extracerebral pressure. Increase in the intracranial pressure *per se,* although generally followed by hernia, is not necessarily the cause of this condition; the increased bulk may be outside the brain itself, as in serous meningitis when, if the dural defect is directly over the serous effusion, no herniation results, whereas if away from it, herniation follows.

In abscess of the brain, hernia cerebri may result from: (1) Increase in the cerebral tissue bulk, viz., the abscess and its surrounding suppuration and reactive changes,—capillary thrombosis, hemorrhages, perivascular infiltration, round-cell infiltration and associated œdema—to which is added, after operation, cerebral death from trauma, secondary reactive œdema, extension or re-filling of abscess, and encephalitis. (2) Obstruction in the exit of the cerebro-spinal fluid from the ventricles. (3) Increase and alteration in the amount of the fluid produced, the alteration probably seriously interfering with exit (such as occurs in the eye in acute secondary glaucoma), although this has not been demonstrated.[2] (4) Strangulation, death, suppuration, and secondary œdema of the hernia itself. By the reaction of these factors, one upon the other, a vicious circle is established.

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[1] Mollison, W. M.: Case of Cerebral Abscess; hernia cerebri; avulsion of abscess wall; complete homolateral ophthalmoplegia; recovery; *Proceedings Roy. Soc. Med.,* London, 1917-1918, Vol. II, Sec. Otol., p. 61.

Post-mortem reports show that in a very large proportion of cases brain abscess is associated with internal hydrocephalus. In an examination of 124 post-mortems of cerebellar abscess internal hydrocephalus was recorded as present in 27 cases. The vast majority of the reports, however, contain no mention whatever of its presence or absence, the association between the two apparently not being recognized. (See Appendix II, Cerebellar Abscess.)

A certain degree of internal hydrocephalus probably accompanies all brain abscesses. The brain abscess and suppurative meningitis experimentally produced by Weed [3] in cats were invariably associated with internal hydrocephalus, although no obstruction existed.[4] The affected hemisphere is frequently displaced beyond the median line, the displacement obstructing one of the main channels of the cerebro-spinal fluid system, thus causing obstructive hydrocephalus. (See p. 129.)

In the production of increased intracranial pressure—an over-filling of the dural envelope—the various factors are so dependent the one upon the other that they should not be considered separately. The intradural suppuration by bacterial, chemical and mechanical irritation induces an associated cerebral œdema and is accompanied by an increased production of cerebro-spinal fluid. At the same time its rapid removal from the interior of the brain is prevented by a mechanical obstruction of the large cerebro-spinal fluid circulatory channels from mechanical displacement of the brain substance—obstructive hydrocephalus,—or from occlusion of the eliminating channels of the cortex, (the subarachnoid spaces)—from the associated meningitis—communicating hydrocephalus.

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[2] Rawling, L. B.: Cerebral Edema (Excess Cerebrospinal Fluid); *Brit. Med. Jour.*, May 4, 1918, p. 499.

[3] Weed, L. H., Wegeforth: Meningitis Produced by Intravenous Inoculation; *Monographs of Rockefeller Institute for Medical Research*, March 25, 1920, No. 12, pp. 57-112.

[4] Dandy, W. E.: Extirpation of Choroid Plexus of Lateral Ventricles in Communicating Hydrocephalus; *Annals of Surgery*, December, 1918, 68, p. 569.

Argot,[5] from observation of forty cases of hernia cerebri following war injuries, contends that infection is always the cause, and that the infection induces a localized or general meningitis, with or without abscess formation, with the production of an excessive amount of cerebro-spinal fluid. He describes a localized meningitis with cyst formation around the dural opening, and contends that if the infection can be combated the hernia will recede.

Argot's observations have been substantiated experimentally by the frequency with which traumatic brain abscess is complicated by subdural abscess situated away from the dural defect.[6]

### CIRCULATORY FACTORS.

Anything that obstructs the return circulation from the head will cause increased intracranial pressure and herniation, if a dural defect exists. The writer has seen ligation of the jugular vein followed immediately by longitudinal sinus thrombosis and intense optic neuritis with total blindness. The blindness, after persisting for several days, was relieved by rupture of the dura and by development of a hernia cerebri. At autopsy the lateral sinus on the opposite side was found to be of very small calibre. The following case illustrates the circulatory disturbances that may follow ligation of the internal jugular vein in sinus thrombosis, with the development of hernia cerebri:[7]

CASE XXIII.

D. S.: Hernia Cerebri. Boy, 9 years.

**Previous History.**—Scarlet fever five years ago, followed by left otitis media purulenta; this subsided and was followed by fair-sized dry central perforation of membrana tympani, which several times had discharged and subsequently became absolutely dry, remaining so for several months at a time.

**Present History.**—Suddenly slight pain, redness, and tenderness over mastoid, accompanied by very profuse discharge. Temperature 100 degrees. Vessels of left optic disc rather full. Following day all mastoid symptoms had disappeared, discharge much less, follicular tonsilitis, although no complaint of throat. Perfectly well in three days. Five days later, sudden dizziness and rise of temperature to 104 degrees. Temperature rapidly fell again to normal, and patient appeared to be perfectly well. Same sudden rise of temperature repeated next day. Following day temperature 104 degrees, which fell in five hours to 100.2 degrees, and rose again in five hours to 103.4 degrees, and again fell in six hours below normal, 98.1 degrees.

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[5] Argot, G.: Pathogénie et Traitement des Hernies Cérébrales; *Lyon Annales de Chirurgie*, 1917, Tome 14, p. 814.

[6] Essick, C. R.: Pathology of Experimental Traumatic Abscess of the Brain; *Archives of Neurology and Pathology*, 1919, Vol. 26, p. 1083.

[7] From *Archives of Otology*, 1906, Vol. XXV, No. 2, p. 91.

Blood examination showed numerous plasmodia malariæ. Quinine given in large doses, but in spite of this there was a further sudden rise on following day.

**Operation.**—Mastoid absolutely normal; no bleeding from diploic veins. Sinus exposed at knee; normal appearance, but blackened well down towards bulb, and small opening in wall very low down through which drop of pus oozed. Jugular ligated; immediately followed by a profuse flow of blood from upper wound, both from soft parts and from bone. On passing probe into opening in sinus, profuse hemorrhage, which was controlled by pressure from above. Free bleeding thought to come from below. Firm tamponage necessary to control hemorrhage.

Following day general condition good, but *marked double optic neuritis, although none had been present one-half hour prior to operation;* chilly sensation, rather restless.

**Two days later:** Temperature 100-103.4 degrees; pain in back of neck on moving eyes; retinal veins enormously dilated and tortuous, arteries small. Numerous hemorrhages in retina. Ether given; again very profuse hemorrhage, supposed to come from below as well as above. Very firm tamponage necessary to control bleeding.

**Four days later:** *Veins over whole of scalp and upper part of chest very much distended.*

**Eleven days later:** General condition very much improved, but can see very little. The enlarged veins in the scalp have felt like irregular cords under the finger, as if filled with firm thrombi, but now are gradually disappearing. Has had two lumbar punctures during past week. Fluid clear, under pressure, microscopical examination negative, first puncture apparently improved vision somewhat. Great difficulty has been experienced in changing dressings because of the hemorrhage which succeeded any disturbance of the firm packing.

**Nineteen days later:** Very free discharge of clear cerebro-spinal fluid, escaping from small opening in inner wall of sinus. Dressings soaked several times daily.

**Twenty-four days later:** Hernia cerebri.

During the next month symptoms of cerebellar abscess, irregular projectile vomiting, vertigo, on left side loss of coördination of both arm and leg, but chiefly of arm. Deviation of tongue, but no loss of flesh.

During this time the cerebellum was twice explored; nothing found.

**Thirty-four days later:** Rapid rise in temperature; delirium; inability to swallow; collapse; death.

**Report of Autopsy.**—"Nothing abnormal noted over the surface of the brain. The optic nerves and vessels cut and the medulla divided and the brain lifted out. It separated easily except at the site of the hernia cerebri, which was the left side of the cerebellum.

"On opening the sinuses, the left lateral, the torcular, the inner one-fourth of the right, nearly the whole of the superior longitudinal, were all thrombosed. The cavernous and the petrosals were normal.

"The hernia cerebri involved nearly the whole of the left lateral lobe of the cerebellum; extending from it was an area of softening involving the left lateral portion of the pons.

"Two specimens were taken for microscopical study; one, the lateral portion of the pons and the other the medulla. The specimen taken from the medulla, on cross-section in its upper part, showed in the median line of the floor of the fourth ventricle a hemorrhagic area extending inwards and slightly to the left. The floor of the fourth ventricle showed microscopically extensive round-celled infiltration. Medulla: both specimens show evidences of rather extensive meningeal inflammation."

Examination of the wet specimen of this dura several months later showed the free right lateral sinus to be only about one-half the size of the thrombosed left lateral sinus.

### SURGICAL PATHOLOGY AND PHYSIOLOGICAL FACTORS.

Hernia cerebri from a surgical standpoint may be divided into two groups: (1) *non-ulcerative,* and (2) *ulcerative or fungating.*

Non-ulcerative hernia cerebri, even in the presence of infection, may serve a useful purpose, as by its development, compression which is favoring an extension of the suppuration may be relieved, the protective forces of the brain being enabled thereby to limit the suppurative process, which otherwise will prove fatal. A non-ulcerating hernia cerebri *is covered* with piarachnoid; it is smooth and glistening, and pial vessels run over it. It occurs with large dural defects. The surgical treatment calls for the preservation of this normal piarachnoid.

Unfortunately, in the presence of suppuration, non-ulcerative hernia cerebri generally becomes ulcerative or fungating. Fungating hernia is not covered with piarachnoid, its surface is ulcerative or gangrenous. It occurs in small dural openings, and is partly the result of strangulation of cerebral tissue.

Fungating hernia cerebri, examined microscopically, is found to contain little normal cerebral tissue, even when the hernia is of large size. In the presence of suppuration a large part of the cerebral tissue protruding from the dural opening rapidly degenerates and is replaced by vascular inflammatory exudate, with the formation of granulation tissue. The area of œdema and granulation tissue passes imperceptibly into the area of normal cerebral tissue, so that if the hernia is excised, the irritation of the operation is sufficient to start the process afresh.[8] Several painful experiences have impressed this upon the writer. Moreover, it is impossible to stop the growth of a hernia by pressure, as the irritation of the compression increases its size.

Normally, the cerebral tissue is bathed in warm, mildly alkaline cerebro-spinal fluid. If a non-ulcerative cerebral hernia is exposed to the air for but a few minutes, its surface becomes dry from evaporation, the pathological fluid of the œdema coagulating rapidly. Drying of the surface of a cerebral hernia results in local areas of gangrene, a frequent and to a large extent unavoidable complication of cerebral hernia. This superficial gangrene continues uninterruptedly whenever any absorbent material is used on the hernia.[9]

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[8] Sargent and Holmes: The Treatment of the Cranial Injuries of Warfare; *Brit. Med. Jour.,* 1915, I, p. 537.

## SURGICAL TREATMENT.

### PREVENTION OF HERNIA; SITE OF DURAL DEFECTION.

Experimental evidence and clinical experience demonstrate that dural defects over the vault of the cerebrum or the lateral lobes of the cerebellum are much more frequently followed by herniation than over the under surface of the temporo-sphenoidal lobe, the inner and lower angle of the forehead, or the anterior surface of the cerebellum, the dura and the brain, at the latter points, being held firmly together by vessels and nerves, and the intracerebral and intracerebellar pressure being minimized. Consequently, drainage should be at the site of the dural defect, unless a protective non-ulcerative hernia is desired for the control of compression.

An effort should be made in every case of hernia cerebri to determine the causative factor or factors. As the underlying cause in many cases of hernia cerebri following abscess formation is an incomplete evacuation of the abscess, if a fistulous tract in the abscess cavity can be located without injury to the cerebral tissue its complete evacuation will be followed by a recession of a large part of the hernia.

(See Case XII, W. K., Chapter IV, p. 60, of Double Cerebellar Abscess, in which hernia, probably from "second abscess," was relieved by evacuation.)

Likewise, the evacuation of a meningeal abscess or retention cyst, situated near the edge of the dural defect,—both frequent complications,—will promptly relieve the hernia.

*The treatment* of hernia cerebri involves the avoidance of irritation from surgical interference or dressings; the protection of the hernia from injury, dryness, cold, or secondary infections; and possibly the reduction of increased intracranial pressure.

It has long been recognized clinically, as previously noted, that excision of the hernia is invariably followed by its reappearance and that, in many cases, it ushers in a fatal meningitis, the rapid increase in the hernia resulting from the mechanical irritation of the operation itself. Surgeons have learned to leave the protruding tissue alone, confining their efforts to attempts at covering the protruding mass. The observation of Essick, Weed and Wegeforth[10] in experimentally produced brain abscess of a fatal meningitis from recrudescence of a latent infection, following operation for the closure of cranial defects, in abscesses which have been healed for several months, explain the complicating meningitis, and should make most cautious the slightest surgical intervention. Even attempts to probe apparent fistulous tracts in the hernia should be avoided, unless instillation with a bismuth solution, followed by X-ray examinations, shows that the fistula leads to a definite cavity within the dural opening.[11]

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[9] Steed, Kellogg: Gunshot Fracture of the Skull; *Jour. A. M. A.;* May 5, 1917, Vol. LXVIII., p. 1299.

[10] *Loc. cit.*

**Protection.**—During exposure for dressing, the hernia should be continually but gently flooded with warm salt solution containing a small quantity of calcium salts. (Cushing has found that sterile normal salt solution without calcium salts is toxic to normal cerebral tissue.) The hernia should be irrigated but infrequently, to avoid all unnecessary irritation of the cerebral tissue and to facilitate the growth of new connective tissue over the hernia.[12] During the war Dakin's solution was used freely on cerebral tissue; its apparent harmlessness must be because the hernia no longer consisted of cerebral tissue, although Argot washes out the cerebral wounds with Dakin's solution immediately after the injury. Cushing [13] instills dichloramin-T in the cerebral tract; its use, however, can be justified only by extensive destruction of cerebral tissue. The writer has seen a hernia become gangrenous with rapidly fatal result, following application to its surface of a weak formalin solution.

After irrigation with normal salt solution, the hernia should be thoroughly covered with sterile wet rubber tissue coated with vaseline, over which a dressing is placed. As the rubber tissue prevents the evaporation of the fluid, it should be changed only at infrequent intervals—not more than once every ten days or two weeks—even though the discharge from contiguous suppuration be irritating to the skin and surrounding parts.

In the case reported (page 135) the odor from the eczema induced in the surrounding skin by the non-changing of the dressings permeated the whole floor of the hospital, but the attendants were not permitted to disturb the hernia unnecessarily. The outer dressing was changed once or twice weekly, and a non-irritating ointment applied to the surrounding skin, but the dressing on the hernia itself was removed only every fourteen days. At these times the hernia was merely flooded with a warm salt solution.

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[11] Tobey, Dr. G. L.: *Personal Communication.*

[12] Adami and McCrae: Text Book of Pathology, 1914, II Edition, p. 124.

[13] Cushing: Notes on Penetrating Wounds of the Brain; *Brit. Med. Jour.*, Feb. 23, 1918, p. 221.

Several months later a small piece of rubber tissue, which had been lost under the osteoplastic flap at the time of the operation, came away followed by a piece of the bone of the flap. Slight recession of the hernia followed; from which it would seem that the irritation from the foreign bodies was responsible, in part at least, for the hernia.

Under non-operative treatment the granulating mass gradually assumes a very even appearance and begins to shrink; later, islands of epidermization appear in it, gradually covering the hernia. (Figs. 31, 32.)

All the cases of hernia cerebri which the writer has known to recover have been treated without operative interference upon the hernia itself.

### REDUCTION OF INCREASED INTRACRANIAL PRESSURE.

Smith,[14] and Sargent and Holmes[15] advise frequent lumbar punctures, using a saline solution manometer to measure the degree of pressure of the cerebro-spinal fluid.

> **Technic.**—"Normally we have found that the column of saline thus supported varies from 15 to 25 cm. The amount of fluid to be withdrawn will roughly depend upon this reading and varies from four to eight fluid drachms, according to the initial pressure recorded. Even where the initial pressure is very high—in one of our cases it amounted to 108.5 cm.—it is not necessary to remove more than from 4 to 8 fluid drachms, as the pressure recorded after the removal of this quantity will rarely register much above the normal. It is important, too, that the fluid be withdrawn slowly, almost drop by drop, as otherwise the hernia, with too much sudden relief of pressure, may sink back through the bony opening into the brain, leaving a deep cavity where before there was a large hernia. This is dangerous, for adhesions may be broken down and a septic meningitis lighted up."[16]

Although the writer has no personal experience with lumbar puncture in cerebral hernia, such a procedure is based upon sound mechanical and clinical grounds provided the hernia is of cerebrospinal fluid origin. The author has seen a marked papillœdema, the result of traumatism, quickly disappear under one lumbar puncture. In another case, following an operation for compound depressed fracture without injury to the dura, the patient, through failure on the part of the medical attendant to appreciate the condition, continued

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[14] Smith, S.: Notes on the Treatment of Hernia Cerebri; *British Medical Journal*, July 22, 1916, p. 103.

[15] Sargent and Holmes: Treatment of Penetrating Wounds of the Skull; *British Journal of Surgery*, 1916, Vol. III, No. 11, p. 475.

[16] Cassidy and Page: A Method of Determining the Absolute Pressure of the Cerebro-spinal Fluid; *Proc. Roy. Med. Soc.*, 1910-1911, Vol. 4, pt. 1, Clinical Section, p. 56.

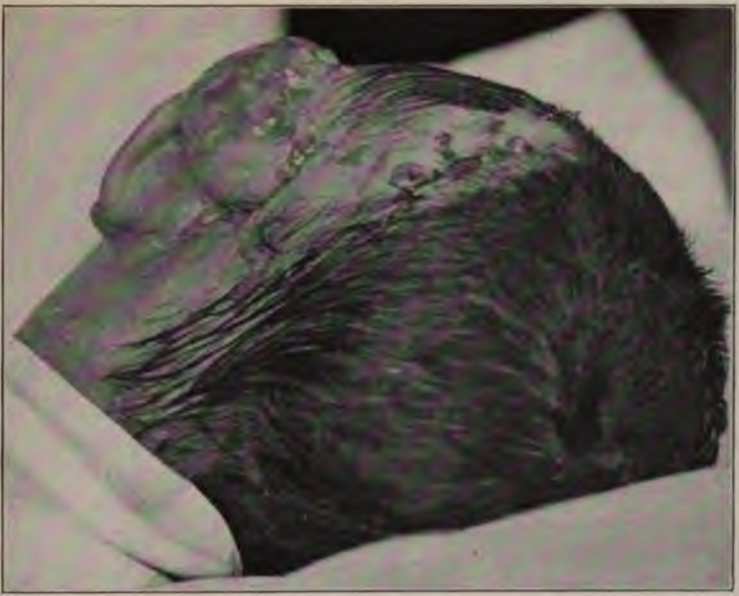

FIG. 31.—Cerebral hernia following evacuation of temporo-sphenoidal lobe abscess. Note outline of osteoplastic flap, all of which is slightly elevated and the lower part of which had been sacrificed. The wide decompression thus furnished was the deciding factor in the recovery. Some months later a small bony sequestrum was spontaneously expelled, but in spite of this loss the patient has a good bony covering over the whole region except just above the ear.

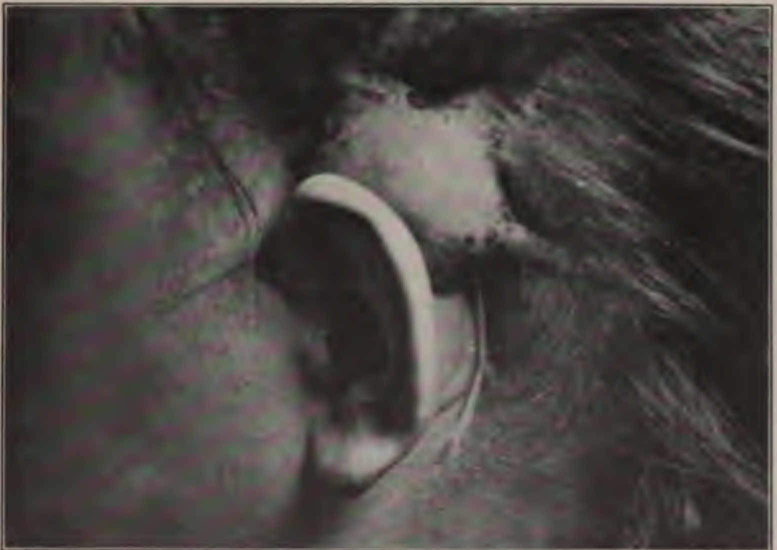

FIG. 32.—The same case as Fig. 31, three years after the evacuation of the abscess, showing recession of hernia and its complete covering by skin without operative intervention or the application of pressure.

in a state of active delirium for three weeks and when seen was apparently dying from exhaustion. One lumbar puncture, however, was followed by almost immediate return to consciousness and rapid recovery. The writer also has seen several cases of fracture with intradural hemorrhage in which each lumbar puncture was followed by marked and immediate improvement of the patient's mental condition. In fact the results of repeated lumbar puncture in traumatic cases have been so satisfactory that with the author it is a routine practice if the operation on the skull is not followed by immediate and continued improvement.[17]

Argot,[18] as the result of his large experience in cerebral hernia, condemns lumbar puncture as being apt to cause a dissemination of the localized infection, meningitis, or rupture of a localized abscess internally.

### CASE XXIV.

J. G.: *Hernia Cerebri Following Operation for Temporo-sphenoidal Abscess.*

Woman. Admitted to hospital December 23, with history of chronic relapsing otitis media for many years. Of nervous disposition; subject to night terror.

Operation on mastoid showed acute exacerbation of chronic process. Discharged January 1, doing well. Hearing: voice, 7 inches; whisper, 3 inches; watch, 2 minutes. Weber referred to left.

Three days later, January 4, became delirious at night, singing, more or less irrational. Re-admitted to hospital the next day. Examination: no nystagmus, could hear with left ear, no spontaneous pointing deviation.

January 6, patient able to walk and execute all movements; irregular contractions over body; active abdominal reflex; no neck stiffness; mastoid wound re-opened; dura over middle fossa found covered with granulations. Sinus accidentally opened; profuse bleeding. The following day there was spontaneous nystagmus on looking towards right side, and aphasia,—naming centre was slightly involved, but the aphasia seemed to be rather a carrying over of a previous impression. Shown a pencil, named it and said it was something to write with; shown a knife, called it a knife; shown a bunch of keys, called it a knife and said it was something to whistle with. Shown a match, she said it was something to light a fire with; shown a pad called it a piece of paper; shown a hospital card, said it was a piece of paper to put your name on.

Convulsive seizures, in which both hands rotated on themselves inward. Lateral deviation of the head and of eyes to the left. The left pupil gradually became much larger than the right, the right apparently becoming much smaller. On coming out of the convulsion undoubtedly had a Babinski, especially on left. The face was drawn to the left as if there were paresis of the right side. As patient became further conscious the left pupil became widely dilated; the right pupil becoming small. At this time there was marked rotary nystagmus to the right. Between convulsive seizures left patellar reflex absent, no hemianopsia. Named objects well for a few minutes and then gradually relapsed into a drowsy condition, repeating one word to everything, although she undoubtedly named the use of things better than the name itself.

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[17] Lobinger, A. S.: Cerebral Œdema in Intracranial Trauma; *California State Journal of Medicine*, June, 1918, Vol. 16, p. 308.

[18] Argot: *Loc. cit.*

Caloric producing nystagmus not associated with any dizziness or vomiting and apparently no pointing deviation, although of this cannot be sure, because of stuporous condition of patient.

January 9. Several convulsive seizures. Right hand cold, left warm; slight double papillœdema, especially on left disc. Toxic delirium. Internal squint, left eye.

**Diagnosis.**—Temporo-sphenoidal lobe abscess, without capsule as length of time since initial cerebral symptoms is too short to allow of formation of capsule.

**Operation.**—Large osteoplastic flap turned upwards. Incision of dura showed cortex to be normal. There was marked prolapse of brain through dural opening. Lumbar puncture was done but resulted in a few drops only of cerebrospinal fluid; canula left in position. Exploration of temporo-sphenoidal lobe was followed by a gush of pus and the immediate free discharge of cerebrospinal fluid from trocar in lumbar region. The greatest delicacy in cleansing abscess. No hemorrhage. Rubber drain. Replacement of flap. Almost tight closure.

February 11. Large hernia about half the size of a tangerine orange directly above ear. Under slight pressure, small amount of pus observed at upper edge. Hernia not gangrenous, kept covered by rubber tissue. Pus seen in two or three places along edge of hernia. Patient apparently perfectly well; able to name objects correctly. Hernia could be reduced by one-third by pressure; it was soft, perfectly smooth, and cystlike in appearance, not like brain; probably composed of granulated or œdematous tissue.

Some weeks later. Hernia, which had been carefully protected, was very much smaller. A piece of rubber tissue was discovered which had been lost during drainage. Loose sequestra of bone removed and hernia gradually reduced. Patient made a complete recovery.

**Comment.**—It was undoubtedly the irritation from the lost drain that caused the hernia.

# CHAPTER IX.

## PROTECTIVE MECHANISM OF THE BRAIN.

The protective mechanism of the brain is one of the functions of the delicate, anatomical and physiological relationship which exists between the cerebro-spinal fluid circulatory system and the circulatory system of the blood, by means of which bacterial invasion is prevented, or, having occurred, the invading toxins are eliminated. The brain is unable to function properly when it is the seat of suppuration; it is also profoundly influenced by toxemia; consequently nature has furnished especial protection both from external infection and from the blood which circulates through it. In addition to the bony cranium an external protection—the dura—is essential, not only because the cranium is liable to injury, but because it contains an organ, the nose, which is the natural habitat of many forms of bacterial life, while another organ, the ear, is the frequent site of infection, both of these being contiguous to the cranial cavity itself. Furthermore, as the blood stream, which circulates through and nourishes the brain, is constantly invaded by micro-organisms (one of its functions being to devour them) there must be a special mechanism whose purpose it is to guard the brain, not only from the organisms themselves, but as far as possible from the influence of the toxins which may be contained within the blood.

**Prevention of Infection.**—Consequently the brain normally is protected from infection from without by (1) the dura; and from within by (2) the normal Encephalo-Chorio-Meningeal Mechanism, consisting of an intimate anatomical and delicately adjusted physiological relationship between the blood circulatory system and the cerebro-spinal fluid circulatory system within the dura, the chief function of which is the nutrition of the brain from the circulating blood (the same as all other tissues of the body). At the same time it affords protection from any mechanical or bacterial irritant that may be circulating within the blood, and from the toxins in waste products discharged into the blood stream again.

In this mechanism the chief protective elements are furnished by (a) the blood in the blood vessels, (b) the blood vessels themselves, (c) the pia and the external layer of the subarachnoid space, (d) the cerebro-spinal fluid, and (e) the glial tissue of the brain.

*Surgical Application:* It is the presence of a special internal protective mechanism which explains the fact that although abscess of the kidneys, skin, etc., is frequent in suppurative endocarditis, abscess of the brain is of very rare occurrence, and then generally as a terminal manifestation, in spite of the fact that the blood infecting micro-organisms have a straight-away course from the heart to the brain.

In the presence of infection the brain repels the invasion (1) by the incorporation of bacteria and deleterious products within the substance of the phagocytic cells, which in the brain substance come from two sources, (a) the leucocytes and (b) the polynuclears from the blood vessels (as in other tissues) and (c) the macrophages from the cerebral tissue itself. These macrophages—a cell peculiar to the nervous system and produced from the external layers of the piarachnoid, the pia and the glia—play a most important part in combating suppuration and in repair. Their chief function, apparently, is (2) the transference and removal of dead and deleterious matter from the central nervous system by incorporating it within their substance and discharging it (a) into the blood vessels and (b) into the perivascular spaces of the cerebro-spinal fluid circulatory system, by which all waste or deleterious material is conveyed into the subarachnoid space, and thence discharged into the venous sinuses or the extracranial lymphatics. During this process there occurs (3) an immunization of the cerebro-spinal fluid in the subarachnoid and perivascular spaces, while at the same time elements are produced from the blood vessels and the brain tissue which (4) limit the suppuration by (a) the production of polymorphonuclear cells (b) the proliferation of glial tissue, and (c) the formation of new connective tissue.

### PATHOLOGICAL PROCESSES IN INFECTION.

Infection enters the brain by four methods:

(1) By the extension of contiguous tissue suppuration, causing a necrosis of the dura, an intrapiarachnoid abscess, a superficial brain necrosis, or a suppurative meningitis;

(2) From the blood vessels, (a) through retrograde thrombophlebitis of the veins of the ear or nasal sinus which perforate the dura and enter the brain, thus causing adjacent (secondary) brain abscess; or retrograde thrombophlebitis of the veins which enter the large venous sinuses from the brain substance, thus causing intercurrent (tertiary) brain abscess; (b) by invasion of the perivascular

channels surrounding the blood vessels—perivasculitis; (c) through bacterial embolus within the vessels—the method of cerebral invasions of all infections that primarily are of blood-stream origin (cerebrospinal meningitis, streptococcæmia with secondary meningitis, pneumococcus meningitis, metastatic abscess from the lungs, etc.);

(3) By invasion of the prolongations of the subarachnoid spaces which surround certain nerves—chiefly the olfactory, the optic, and the auditory nerves—by means of which the subarachnoid spaces become extracerebral. (The filtrable virus of poliomyelitis probably enters the nervous system from the nasal mucosa by way of the perivascular prolongations which surround the olfactory nerves);

(4) By interference with the normal operation of the meningeal protective mechanism, which, in the presence of blood-stream infection, may allow entrance into the meninges, the perivascular spaces, or the cerebral substance itself, of micro-organisms from the blood stream. This latter method probably is a frequent factor in the production of streptococcus meningitis and pneumococcus meningitis of aural and nasal accessory sinus origin.

## PROTECTIVE MECHANISM OF THE BRAIN.

### I.—By the Dura.

Surgically the chief function of the dura is to prevent the entrance of micro-organisms into the brain. On the approach of an adjacent infection the dura fortifies itself and combats the poison by the formation of granulation and subsequently of new connective tissue, both on its external and internal surface—a pachymeningitis externa and interna. In the development of the latter process the central protective mechanism, especially the arachnoid cells, takes an active part.

*Points of Vulnerability of the Dura*: Although the enclosing dura is the chief protection of the brain against direct invasion by micro-organisms, it contains areas of vulnerability, especially where it is perforated by certain nerves and blood vessels. The subarachnoid space prolongations surrounding these blood vesels extend well into the foramina through which the nerves and blood vessels enter and leave the cranium. At these sites the subarachnoid space actually becomes intracranial. The subarachnoid prolongation around the nerves causes a perineural space in three places; (1) along the nerve of the special sense of hearing and orientation, where the intralabyrinthine fluid is part of the cerebro-spinal fluid; (2) along the nerve of the special sense of smell—the olfactory nerve—where the perineural sheaths descend into the nasal mucous membrane, and (3) along the nerves of the special sense of sight, where the subarachnoid space is continued for a certain distance along the optic nerve as the intervaginal space.

It is more than a coincidence that all these prolongations of the subarachnoid space are directly connected with three of the organs of special sense. There is experimental evidence that it is through the extradural or extracranial prolongation of the perivascular spaces of the vessels and of the perineural spaces of the nerves that infection frequently enters the brain, and that within these recesses micro-organisms long continue to persist after their elimination from the general subarachnoid space.

### II—By the Subdural Space.—

The subdural space apparently is very immune to infection, as it offers especial resistance to the growth of bacteria. In meningitis or brain abscess it generally is unaffected, although as a brain abscess approaches the cortex the subdural space may become completely obliterated; but in cases of pachymeningitis interna the infecting micro-organisms may project well into the subdural space without involving it, as demonstrated by Streit.[1] In this situation they may remain more or less dormant for long periods of time. If trauma is now added, breaking down the adhesions, a general fulminating meningitis may follow, as has occurred in many cases following an operation for a chronic aural or nasal sinus suppuration.

*Surgical Application:* Although meningitis or abscess primarily confined to the subdural space is a very rare occurrence from aural infection,[2] it apparently is more common in nasal sinus suppuration, probably because of the communication of the subdural space with the mucosa of the frontal sinus by way of the perimeningeal spaces.[3] However, in experimentally produced traumatic abscess,[4] large subdural abscesses were frequent, and microscopic examination found the adjacent subarachnoid space to be free from micro-organisms, demonstrating the extreme degree of resistance of the external layer of the arachnoid and explaining the clinical experience that a subdural abscess is a frequent complication of hernia cerebri following evacuation of an intracerebral abscess. The occurrence of a hernia should always awaken suspicion of its origin in a localized collection of pus in the subdural space, and should cause the surgeon carefully to expose the regions adjacent to the hernia before again entering the brain surgically.

### III—By the Subarachnoid Spaces.—

In the normal state the subarachnoid spaces, in which the cerebro-spinal fluid circulates, are kept free. On the approach of bacteria to the dura the subarachnoid space fortifies itself by an overproduction of cerebro-spinal fluid containing a large number of phagocytic cells—polymorphonuclears.

*Surgical Application:* Serous meningitis is a protective process. In aseptic meningitis the perivascular spaces apparently are not involved, but the velum interpositum and choroidal stroma are infiltrated with polymorphonuclears. The high polymorphonuclear count without organisms in lumbar puncture is simply a sign of a protective meningitis. Later, as the process becomes quiet, mononuclear cells and lymphocytes predominate.[5]

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[1] Streit, Hermann: Weitere Beiträge zur Histologie und Pathologie der Meningitis und Sinusthrombose; *Archiv für Ohrenheilkunde,* 1912, Bd. 89, S. 177.

[2] Case VIII, Chapter Four, p. 37.

[3] Zwillinger, H.: Experimentelle Untersuchungen zur Mechanik der intrakraniellen und cerebralen Komplikationen der Stirnhöhlenentzündungen. (From the Anatomical Institute of the University of Budapest.) *Archiv für Laryngologie und Rhinologie,* 1914, Bd. XXVIII, S. 271.

[4] Essick, Charles R.: *Loc. cit.*

On the entrance of bacteria into the subarachnoid spaces of the cerebro-spinal fluid circulatory system, the meningeal protective mechanism combats infection chiefly by the action of macrophages coming from the glial tissue and from the piarachnoid cells.

## ANATOMY OF THE CEREBRO-SPINAL FLUID SYSTEM.[6]

The cerebrospinal fluid circulates within closed cavities and tubes—the ventricles, the cisterna and the subarachnoid spaces constituting the cerebro-spinal circulatory system—just as the blood circulates in the blood vascular system; and like the walls of the venous system, the walls of the cerebro-spinal circulatory system are normally impermeable to the contained fluid.

The cerebro-spinal fluid finally empties into the blood stream after performing its functions in the brain, passing by osmosis into the venous sinuses of the head, and at certain points where the system has passed outside of the cranium, communicating again by osmosis, with the extracranial lymphatic system. This communication occurs along the arachnoid prolongations around the sheaths of certain nerves, especially the olfactory and the auditory.

Thus, while the cerebro-spinal fluid circulatory system is a closed system without an apparatus for the purification and return of its fluid such as the vascular system has in the heart and lungs, it utilizes both the vascular and the lymphatic system for its discarded material much in the same way as the blood vascular system utilizes the kidneys; with this difference, however, that while the blood discharges through the kidneys only its deleterious material the entire cerebro-spinal fluid constantly is being discharged into the blood or the lymphatic system.

Viewed then in a large way, both anatomically and physiologically, the cerebro-spinal fluid circulatory system must be of importance to the cerebral functions.[7 to 16]

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[5] Ayer, J. B.: *Loc. cit.*

[6] Eagleton, W. P.: Operative Treatment of Suppurative Meningitis with special reference to Irrigation of the Cranial and Spinal Subarachnoid Spaces, and the Importance of Protective Meningitis from a Prognostic and Therapeutic Standpoint; Transactions American Otological Society, 1921.

[7] Weed, Lewis H.: Studies on Cerebro-Spinal Fluid; *Journal of Medical Research*, September, 1914, Vol. XXXI, pp. 21-117.

[8] Wegeforth, Paul, and Weed, Lewis H.: Studies on Cerebro-Spinal Fluid; *Journal of Medical Research*, September, 1914, Vol. XXXI, pp. 167-176.

[9] Weed, Lewis H., and Cushing, Harvey: Studies on Cerebro-Spinal Fluid; *American Journal of Physiology*, January, 1915, Vol. XXXVI, No. 2.

[10] Cushing, Harvey, and Weed, Lewis H.: Studies on the Cerebro-Spinal Fluid and its Pathways; *Johns Hopkins Hospital Bulletin*, November, 1915, Vol. XXVI, No. 297.

The circulating cerebro-spinal fluid is produced chiefly from the choroid plexus within the ventricles, the walls of which are lined by cells, the endyma, which are impervious to the fluid. From the ventricles the fluid passes by a process of overflow through the aqueduct of Sylvius into the fourth ventricle and leaves it by the medial foramen of Magendie and the lateral foramen of Luschka; from these it spreads over the base of the brain, accumulating in the large basal cisterna, whence it is distributed by way of the sponge-like, communicating meshes of the subarachnoid spaces over the cortex, through the vessels and convolutions, and empties at last by osmosis into the venous sinuses.

In its passage over the cortex there is contributed from the cerebral substance—through the prolongations of the subarachnoid spaces along the vessels entering or leaving the cerebral tissue, called the perivascular spaces—a small amount of cerebrospinal fluid which has a somewhat different composition from the cerebro-spinal fluid formed in the ventricles. These perivascular channels opening directly into the subarachnoid spaces of the cortex, are undoubtedly the means of direct communication between the intracellular juices of the brain cells—the perineural system, and the general cerebrospinal circulatory system.

The cerebro-spinal fluid acts to remove the waste products of the brain, but whether it supplies any nutritive ingredient necessary to the continued functioning of the brain is not known. It is inconceivable that it does not possess some such quality, especially since its normal content is a sugar-reacting substance.

### Impregnability to Infection of Cerebro-spinal Fluid System.

The cerebro-spinal fluid system, while anatomically composed of very delicate cells, is very resistant to infection when attacked either upon its external surface, or internally from the cerebral tissue; and while in close anatomical proximity to the blood circulatory system—being separated from the pial vessels by only one layer of cells and actually surrounding the vessels as perivascular

[11] Weed, Lewis H.: The Formation of the Cranial Subarachnoid Spaces; *The Anatomical Record*, May, 1916, Vol. X, No. 7.

[12] Weed, Lewis H.: An Anatomical Consideration of the Cerebro-Spinal Fluid; *The Anatomical Record*, May, 1917, Vol. XII, No. 4.

[13] Weed, Lewis H., and McKibben, Paul S.: Pressure Changes in the Cerebro-Spinal Fluid Following Intra-Venous Injection of Solutions of Various Concentrations; *American Journal of Physiology*, May, 1919, Vol. XLVIII, No. 4.

[14] Weed, Lewis H., and McKibben, Paul S.: Experimental Alterations of Brain Bulk; *American Journal of Physiology*, May, 1919, Vol. XLVIII, No. 4.

[15] Weed, Lewis H.: The Cells of the Arachnoid; *Johns Hopkins Hospital Bulletin*, October, 1920, Vol. XXXI, No. 356.

[16] Macklin, Charles Clifford, and Macklin, Madge Thurlow: A Study of Brain Repair in the Rat by the Use of Trypan Blue; *Archives of Neurology and Psychiatry*, April, 1920, Vol. III, pp. 353-394.

spaces—the cerebro-spinal fluid system, when uninjured, does not allow of invasion of the subarachnoid spaces by bacteria circulating free in the blood stream. However, a very small disturbance of the meningeal protective mechanism in the presence of a blood-stream infection may allow the invasion of the meninges from the blood stream.

The subarachnoid space bacteriologically resembles the peritoneum, each being a mesothelial-lined cavity which, on account of its blood supply and its contained fluid, furnishes a favorable soil for the development of micro-organisms. The cells of the subarachnoid space, however, both of the external layer toward the subdural space and of the pial layer, are very resistant to infection; but the cells lining the cavity itself possess much less resistance if once infected. Likewise there is experimental evidence that the cerebro-spinal fluid normally possesses to a high degree immunizing bodies which under favorable circumstances may considerably increase. On the other hand, anything which alters the integrity of the meningeal choroid protection, or upsets the operation of the meningeal mechanism, diminishes or removes its protective function.

In suppurative processes the arachnoid membrane thus serves to limit the spread of infection from the outside into the subarachnoid space, or, when the subarachnoid space itself is infected, it prevents infection from spreading inward into the deeper layers of the pia and the substance of the brain. In abscess of the brain, although the perivascular spaces may be filled with exudate, no micro-organisms and but few cells may be found in the subarachnoid space at a distance from the infection.

*Surgical Application:* It is the resistance of the piarachnoid to invasion from the subarachnoid space that explains the clinical fact that while suppurative meningitis both local and general, frequently follows intracerebral abscess, localized meningitis never is followed by the latter, although frequently accompanied by a superficial brain necrosis.

Note: The cerebro-spinal circulatory system seems to have resisting power to infection in itself, as the infiltrable virus of poliomyelitis—which apparently finds its path into the venous system by the nasal mucous membrane—probably in a normal state is prevented from entering the brain by the meningeal mechanism; but microscopical changes of the meningeal mechanism, such as the production of an aseptic meningitis by the injection of a normal horse serum or a normal salt solution—Ringer's or Locke's solution—or slight hemorrhages which have a demonstrable effect upon the meningeal mechanism, "suffice to remove the power of the intact organism to exclude the virus of poliomyelitis from the interstices of the central nervous tissues." [17]

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[17] Flexner, S., and Amoss, Harold L.: Relation of the Meninges and Choroid Plexus to Poliomyelitic Infection; *Journal Experimental Medicine*, 1917, Vol. 25, p. 525.

### INFECTION OF CEREBRO-SPINAL FLUID SYSTEM FROM BLOOD STREAM.

Normally infection is prevented from reaching the brain through the blood vessels by two factors: (1) the intima of the blood vessels of the brain, and (2) the encephalo-meningeal protective mechanism, which includes the choroid plexus.

Given an injury to the intima—a toxemia causing degeneration, or a traumatic injury—a thrombus develops which interferes with the nutrition of the cerebral tissue and, being septic, implants micro-organisms in the already damaged cerebral tissues. Again, without thrombosis, if the meningeal protective mechanism itself is upset—as by lumbar puncture, compression of the jugular, cessation of the heart,—in the presence of pathognomic micro-organisms free in the blood stream at the time of the disturbance, certain micro-organisms may enter the leptomeningeal spaces in sufficient numbers to occasion a fatal leptomeningitis.[18]

### BRAIN ABSCESS FROM MENINGITIS.

In all the experimentally produced meningitis, only one brain abscess occurred —this a superficial one—showing that it is not the micro-organisms in the blood stream that causes a brain abscess—there must be another factor added to this. This factor is undoubtedly thrombosis of a vessel, and the affinity of certain micro-organisms for cerebral tissues.

CASE XXV.

F. V.: *Case of Streptococcus Meningitis from Disturbance of the Meningeal Protective Mechanism of the Brain by Lumbar Puncture in the Presence of a Blood-stream Infection from a Suppurative Thrombophlebitis of the Leg.* Male. Entered hospital October 3, with chief complaint of pain in left leg and groin, which caused inability to walk, sleeplessness and slight headache.

**History.**—Present illness dates from a week before admission to hospital, when he received a blow on the head—in left parietal region. He was not unconscious and worked next day. The following day felt better. Three days before admission he began to have severe pain in left thigh and groin. Past history negative.

**Physical Examination.**—Patient did not look ill; head showed no evidence of injury; eyes, nose and ears, negative; slight tenderness over left hip; knee jerks diminished; no paralysis or sensory changes. There were two eye-ground reports, one that the discs were indistinct, the other that they were clear. X-ray for fracture of skull and enlargement of sella turcica both negative; for left hip injury, negative. Cell count, 18,000; polynuclears, 76%; lymphocytes, 24%. Temperature, 102°.

*October 4*—(Next day)—Lumbar puncture; spinal fluid clear, transparent, no nebula; globulin, negative; cell count, 3 per cmm. That evening temperature began to rise, thereafter running a typically septic course, ranging between 100° and 105°.

The after-history of the case is typically a septicemia. Another lumbar puncture was performed October 14, which showed markedly bloody fluid, containing pus cells and numerous chains of streptococci.

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[18] Weed, Lewis H.: Meningitis Produced by Intra-Venous Inoculation; *Monographs of Rockefeller Institute for Medical Research*, No. 12, March 25, 1920, pp. 57-112.

*October 15*—Patient became delirious and remained so to the end.

*October 24*—Death.

**Autopsy:**

**Head.**—Brain covered by plastic, purulent meningitis, having greenish-yellow look; also a slight encephalitis; sinus free and clear; no fracture to be seen.

**Abdomen.**—Liver slightly enlarged; all organs in abdomen show acute cloudy swelling; inferior vena cava from level of fourth lumbar vertebra down, and extending into iliacs and femorals on both sides, was the seat of a purulent, septic thrombosis; at most central part of involvement was an organizing clot; there was an involvement of the tissues surrounding the vessels.

**Chest.**—Acute, cloudy swelling of heart, and œdema of lungs, with congestion of bases.

**Probable Cause of Death.**—Suppurative meningitis (streptococcus), with original focus a suppurative thrombophlebitis.

**Secondary, or Terminal Lesions.**—Acute, cloudy swelling of heart muscle, liver and spleen; pulmonary parenchymatous nephritis.

### ELIMINATION OF BACTERIA FROM THE SUBARACHNOID SPACE.

While the protective mechanism of the brain prevents the entrance of bacteria into it from the blood stream, when the subarachnoid space has actually been invaded by them the protective mechanism avails itself of the blood stream for their elimination.

Experimental injections of bacteria into the subarachnoid space invariably are followed by finding the micro-organisms in the blood stream.[19] Experimental injection into the subdural spaces of large amounts of normal solution, on the other hand, shows no changes in the brain, but a severe hyperemia of both lungs, with hemorrhages.

*Surgical Application:* Patients in coma or convulsions frequently have a complicating pneumonia, due to a lowering of the resisting power.

**Immunization.**—Immunization in general is so imperfectly understood—especially as comparatively few experiments in this field have been made—that little real knowledge is to be expected regarding its application to the brain and subarachnoid spaces. It is known, however, that full blood contains components which retard the growth of bacteria and in the majority of cases kill them. By this means the blood stream constantly purifies itself. It is also known that the removal of immunized blood from the body allows a continuation of these components for varying times for different micro-organisms. After the third day, however, these component elements of the blood rapidly disappear.

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[19] Felton, L. D., and Wegeforth, Paul: The Production of Experimental Meningitis by Direct Inoculation into the Subarachnoid Space; *Monographs of Rockefeller Institute for Medical Research,* March 25, 1920, No. 12, p. 7.

It has been proved experimentally that immunization of the cerebro-spinal fluid is much more difficult than immunization of the blood, because the cerebro-spinal fluid, being constantly emptied into the venous circulation, cannot very long contain the immunizing substances. Sera injected into the cerebro-spinal fluid are passed into the blood in about twenty minutes.

***Surgical Application:*** It is an established surgical fact that the dura can be incised with greater immunity through the area of a neighboring infection—over the tegmen, for instance—than over an area which is not the seat of an infection. Immunization probably exists in the cerebro-spinal fluid and in the brain area, but the immunization does not extend far nor does it last long. Under such circumstances, the surgeon cannot with impunity enter the dura and explore the brain for any great distance. By so doing he carries infection into an area that is not immunized, while the trauma of the exploration kills cerebral tissues which furnished a most favorable soil for the development of cerebral suppuration.

### SUSCEPTIBILITY OF CEREBRAL TISSUE TO BACTERIAL VIRULENCE.

The ordinary pathogenic micro-organisms are from forty to two hundred times more virulent in the subarachnoid space than in any other part of the body structure. The toxins of tubercle bacillus, for instance, apparently have a specific affinity for meningeal tissue.

Extracts of cholera bacillus are about forty times more toxic in the subarachnoid space than when injected into the blood. Anthrax extract produces hemorrhage into the pia, while many other forms of infection—such as streptococcus capsulatus, or pneumococcus type three—have a distinct action on the blood, causing hemorrhage into the brain.

Newborn infants are subject to certain infections to which adults are less liable, or not at all susceptible. During infancy the colon bacillus is a frequent cause of infection of the brain tissue and of the meninges as well as of the kidneys. Artificially fed infants are much more liable to infection by the colon bacillus than are breast-fed infants. In adults the colon bacillus is only feebly virulent. It would seem that infants during the nursing periods acquire an immunity from the mother. When deprived of this immunity they must build up immunity for themselves.

Bull's[30] experiments on dogs and rabbits demonstrated the pathological effect on the nervous system of streptococci from the blood stream. In dogs sections of the central nervous organs showed multiple abscesses in the cerebrum, the pons, medulla and the white matter of the spinal cord. In some of the rabbits obvious clinical symptoms were wanting, but a purulent meningitis was found at autopsy. The sections showed at times focal lesions associated with the meningitis, usually affecting the cerebrum, the cerebellum, and medulla and the pons.

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[30] Bull, Carroll G.: Pathologic Effects of Streptococci from Cases of Poliomyelitis and Other Sources; *Journal of Experimental Medicine*, 1917, Vol. XXV, p. 557.

### SENSITIVENESS OF BRAIN TO TOXIC INFLUENCES.

To function properly the brain must be bathed in its own normal fluid and at a normal temperature. It is not only highly sensitive to toxic influences, but it is profoundly influenced by any change in the composition of the cerebro-spinal fluid which surrounds it; even sterile normal salt solution (if it does not contain a small amount of calcium salts) is highly toxic to the brain, causing irritation if injected into the subarachnoid space, and respiratory embarrassment and death if irrigated through the cerebro-spinal fluid system.[21]

*Note:* "Changes in the structure or function of the meningeal-choroid plexus complex too slight to be detected by cellular or chemical changes in the cerebrospinal fluid, or by morphological alterations, are sufficient to diminish in an essential manner its protective powers. An immune serum injected intra-spinally is protective, but the degree of its efficiency is more or less proportional to the freedom from injury to the meningeal structure by the irritating substances employed. The slighter the inflammation the more readily and quickly the injury is repaired," and consequently, "Aseptic fluids which irritate, inflame, or even slightly alter the integrity of the meninges and choroid plexus, diminish or remove their protective function." "Injury to the blood vessels of the meninges also promotes an infection. This may be due either to the escape of the inoculated virus from the blood through the injured vessels into the meninges, or, what is more probable, to the entrance of the blood into the subarachnoid space, thereby setting up a mild inflammatory reaction which suffices to promote the infection."[22]

### PROTECTIVE MECHANISM IN THE BRAIN ITSELF.

The protective mechanism in the brain itself is made up of two parts—(1) those structures which prevent the entrance of microorganisms into the cerebral tissue and (2) those forces which combat, destroy, or limit infection which has gained entrance. They frequently act in both capacities. The chief protective agent in the brain in combating infection is the blood, the blood vessels furnishing the elements which defend the cerebral substance from infection. Consequently, the cortex, which is well supplied with blood, is infrequently infected, while the deep substance of the brain, relatively poorly supplied with terminal blood, may easily become the seat of an abscess. In the presence of infection, infiltration of blood into the substance of the cerebral tissue undoubtedly favors the extension of the infection, probably by causing death of the cerebral tissue by pressure.

[21] Weed, Lewis H., and Wegeforth; *Loc. cit.*

[22] Flexner, S., and Amoss, Harold L.: Relation of the Meninges and Choroid Plexus to Poliomyelitic Infection; *Journal of Experimental Medicine*, 1917, Vol. 25, p. 525.

*Note:* There is experimental evidence that the red corpuscles themselves help to limit the extension of infection in certain parts of the brain and under certain conditions, probably by some form of mechanical protection. "A large intra-arachnoid space hemorrhage completely protected the subarachnoid space from infection, although the brain substance was one large abscess." Free red blood cells are also found in considerable numbers in the necrobiotic zone of intracerebral abscess.[23]

### VARYING DEGREES OF SENSITIVENESS TO INFECTION.

The different elements of the brain possess relatively different degrees of resistance to infection. The gray matter of both the cortex and the central ganglia is very resistant. In traumatic abscess the different zones of this gray matter also differ in their susceptibility. An intracerebral abscess in its growth usually does not affect the ganglionic tract, but follows the white fiber tracts.

### LIMITATION OF SUPPURATION WITHIN THE BRAIN.

The blood vessels furnish the elements which encapsulate a suppuration, while by the blood are contributed the phagocytes which help to remove the products of tissue disorganization. The cerebral tissues—the arachnoid, the pia and glia—furnish the macrophages which by a process of digestion assimilate into themselves the dead tissue and carry it to the blood vessels and cerebro-spinal fluid system, that remove it from the brain. In this process the perivascular spaces of the blood vessels play a large part; consequently, in all suppurative lesions of the brain the adjacent perivascular spaces are found crowded with round cells and with micro-organisms.

*Note:* For a detailed description of how a suppurative process into the brain is limited, reference is made to Part II, page 47.

### EXTENSION OF INFECTION.

The three great factors in the death of cerebral tissue, and consequently, in the development of a cerebral abscess are (1) thrombosis, (2) compression, and (3) operative trauma.

After encapsulation, a brain abscess extends by two processes, (1) a seeping of infected material through the capsule and (2) an extension through pressure erosion.

Extension of infection within the cerebral tissue is favored by death of cerebral tissue largely because the myelin discharged from its sheath furnishes a favorable soil for the growth of bacteria.

The blood vessels play such an important part in the proper function of the brain that any disturbance of the circulation in the presence of infection, favors extension. Thrombosis of the blood vessels furnishes a large part of the pathological picture in the early stages of brain abscess. In acute abscess the first extension of the abscess is entirely through thrombosis; consequently, anything which causes dislocation of the brain mass, favors thrombosis and prevents the reactive forces of the brain from operating properly. Compression by favoring thrombosis has a similar action: thus, with an intact dura, dilatation of the ventricles by increase of the intracranial pressure is an aid to extension of the infection.

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[23] Essick, Charles R.: *Loc. cit.*

*Surgical Application:* In the early stages of brain abscess relief of intracranial pressure by lumbar puncture undoubtedly assists in the limiting of the process, as the brain's bulk is primarily not seriously increased, the abscess being due to nutritional death. It is the reactive forces of the brain itself that occasion a part of the increase of the intracranial pressure.

### REMOVAL OF DEAD TISSUE.

Dead tissue within the brain is taken care of by two processes: (1) removal, chiefly by the phagocyte cells of the brain itself—the macrophages—and (2) encapsulation of the dead area. In the former process the same exhaustion of reaction happens in the brain that happens with other tissues; namely, if suppuration is long continued a limited area of suppuration is allowed to exist indefinitely. Experiments with vital dyes show that the macrophages are able to remove a certain amount of dead tissue, but, after a time they lose their activity.

*Surgical Application:* The surgeon must guard, therefore, against allowing the activity of the protective elements to become exhausted. Aseptic meningitis is dangerous, although it contains no septic factors, because it exhausts the protective elements in their effort to remove the cause of irritation.

**Repair.**—Repair of cerebral substance is accomplished by one of two processes: (1) by cyst formation, if the lost tissue is adjacent to the meninges, the pia filling the gap. This accounts for the frequency of meningeal cysts in areas injured. The cysts are filled with cerebro-spinal fluid and the degenerated elements of the blood cells; and (2) by proliferation of glial tissue—a gliosis.

**Recrudescence of Latent Infection.**—Recrudescence of latent infection is a very frequent occurrence in the nervous system. This is of the greatest surgical importance because, a brain abscess having been cured, there undoubtedly lurk in parts bacterial elements which await opportunity again to become active;[24] consequently, no further surgical interference should be undertaken at least for a considerable period of time.

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[24] Eagleton, W. P.: Meningitis, Case 8, *Tr. Am. Otological Soc.*, 1921, p. 919.

Weed and Wegeforth,[25] after curing experimental brain abscess, found that meningitis resulted on further manipulation in trying to close the dural defect.

**Conclusions.**—It would seem then, that it is the close and delicate relationship between the cerebro-spinal fluid circulatory system and the blood circulatory system, which furnishes the chief mechanism in the protection of the brain substance. Both are necessary for the prevention, combating, limiting, and removal of deleterious matter from the brain into the subarachnoid spaces and so out into the circulation. Anything, therefore, which in the presence of micro-organisms interferes with the capillary blood stream within the brain, renders an infection of the brain liable, while anything, operative or therapeutic, which upsets the cerebro-spinal fluid circulatory system increases the likelihood of cerebral abscess or meningitis. In abscess of the brain the chief causative influence in infection is thrombosis of the small vessels; for in consequence of the presence of small thrombi an area of cerebral substance not only undergoes nutritional death but, because of the accompanying perivasculitis, it has eliminated from it the avenue whereby infective material is removed from the brain. Thus, both the blood stream and the cerebro-spinal fluid circulatory system are thrown out of action in this area, while the dead cerebral tissue becomes a favorable soil for the growth of micro-organisms.

### RELATIONSHIP BETWEEN SUPPURATIVE MENINGITIS WITH BRAIN ABSCESS AND PROTECTIVE MENINGITIS.

Although suppurative meningitis is usually the terminal process of brain abscess, it is an important fact that with micro-organisms free in the spinal fluid (as shown by lumbar puncture) an existing brain abscess is the only type which offers a probable chance of recovery, because the protective character of the process which originated from the abscess was already active at the time of the invasion of the cerebro-spinal fluid by the bacteria. Of all the cures of true suppurative meningitis, (not of blood stream origin, reported in the literature)—27 in number, over two-thirds had a protective meningitis at the time of the invasion of the subarachnoid space by the bacteria; of these, five were secondary to a brain abscess and were cured by evacuation of the abscess, together with lumbar puncture or the injection of serum into the spinal cavity.

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[25] Weed and Wegeforth: *Loc. cit.*

"Two were preceded by brain abscess—Day: One was associated with intrapiarachnoid abscess—Held & Kopetsky; and two were associated with or preceded by subdural abscess (traumatic), Kostlivy & Poirier."[26]

It should be recognized that the protective meningitis which develops to ward off the brain abscess from the subarachnoid space may be the deciding factor in controlling the infection when the micro-organisms actually invaded the cerebro-spinal fluid system itself. Consequently, in suppurative meningitis, complicating a brain abscess, in spite of the presence of the invading micro-organisms in the cerebro-spinal fluid, evacuation of the abscess should be undertaken with a fair prospect of recovery from the meningitis.

**CASE XXVI.**

T. P.: *General Suppurative Meningitis* (Streptococcic, Hemolytic) *originated from a latent and unsuspected cerebral abscess of eighteen months' duration* treated by cerebral and cerebro-spinal subarachnoid irrigation.

**Résumé of Case:** About one and a half years previously the patient attempted suicide by shooting himself in the right temple with a revolver. He was brought to the hospital and operated upon by the author who removed a pistol bullet from the left parietal region, the ball having passed through both frontal lobes. The X-ray after the operation showed small fragments of bone or bullet near the wound of entrance, but the patient made a very good recovery and apparently has been quite well since. It is interesting to note, however, that after this time his history states that he has become a drug addict.

A few days before his death, the patient was found by the police in a more or less dazed condition and brought to the City Hospital, where he was placed in the observation ward for psychopathics, little attention being paid to his former cerebral injury. It was noticed that he had a stiff neck, the patient walking around the ward in this condition. The interne performed a lumbar puncture which showed a cloudy fluid under pressure; with numerous pus cells and many Gram positive cocci, many arranged in chains. A diagnosis of streptococcic suppurative meningitis was made.

Two attempts were made to wash out the subarachnoid cerebral space, and anti-meningococcic serum was given intraspinously. The patient grew worse after the second operation, but had appeared in much better condition after the first. Post-mortem disclosed three latent abscesses, the oldest of which doubtless was present from the time of the original injury eighteen months previous. One abscess had perforated the lateral ventricle and slowly discharged its contents into it. From this the infection had passed to the basal cisterna and the cerebellar surface, the subarachnoid spaces of the cortex remaining unaffected.

**History:** June 7, admitted to City Hospital. Temperature 100.2 degrees and pulse 100.

Psychiatric Examination, Wednesday, June 8: "I've been here three or four weeks." (Was admitted yesterday.) Does not know day, month or year. Says "1821." At 10 a. m. says it is about 3 o'clock. "I don't know why I am here. I don't know who brought me here, either. I wasn't drunk. I don't know what was the matter with me."

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[26] Eagleton, W. P.: Operative Treatment of Suppurative Meningitis with special Reference to Irrigation of the Cranial and Spinal Subarachnoid Spaces, and the Importance of Protective Meningitis from a Prognostic and Therapeutic Standpoint; *Transactions American Otological Society,* 1921.

Why do you stay here? "I don't know that, either." Using drugs? "No." Anything hurt you? "No." Were you here before? "I imagine so." Have any trouble? "We had quite an argument—my brother—a little family affair." Did the police bring you in? "I don't know." Did you try to kill yourself? "No."

"Harding is President; Wilson before him."

"4 × 4 = 16 + 10 = 26 — 8 = —well—" (Then no answer).

"9 × 9 = 81 + 9 = 81 —oh, well—" (Then no answer).

"6 × 6 = 36 + 8 = —well—" (Then no answer).

"20 — 1 = 19." (Correct, but slow).

"Before July comes August; before December, November; before October, September—" (after some thought).

Name the months of the year. "This month—" (Then no answer).

Memory for numbers poor—"3, 4, 8, 7."

No dysarthria.

Both pupils very sluggish to light—both irregular. Suspicion of Romberg. Knee jerks exaggerated. Urine dribbled during examination. Executes commands sluggishly. Gait somewhat spastic. Marked tremor of fingers.

*June 9:* Incontinent; neck stiff.

**Lumbar Puncture.**—Fluid cloudy under slight pressure; Wassermann negative; direct smears show streptococci.

**Operation.**—Cerebral and spinal subarachnoid irrigation. Three trephine openings made in right fronto-parietal region; in the first it was found impossible to enter fluid because of apparent adhesion of dura to brain, although the opening was away from the skull opening through which the ball had been extracted, by at least one and one-half inches. Two other openings were then made: opened into the subarachnoid space, and tap made into cisterna magna. Needle inserted underneath dura and arachnoid and surface of brain flushed out with Ringer's solution and drained through needle in cisterna. Process repeated twice through trephine one inch posterior to first opening on the same side. Later the excess fluids were drained through a lumbar puncture. Flaps closed with black silk.

*June 11:* Temperature 99 degrees; pulse 96. Nurse's Note: "Patient appears to be much better. Answers intelligently to questions. Neck still very rigid and head thrown back."

Lumbar puncture; 28 c.c. withdrawn, fluid cloudy, slightly yellow, under slight pressure. Anti-meningococcic serum 20 cubic centimeters injected. At this time the author examined the patient; the man was in excellent condition. He recalled all about the previous experiences in the hospital eighteen months ago.

6 p. m.: Second subarachnoid irrigation. Anterior flap opened; stitches removed; needle inserted into arachnoid and irrigation carried on as on the previous day. Drained through cisterna and lumbar puncture. Fluid yellow and somewhat turbid.

8 a. m.: Answered when spoken to. Pulse fair.

8:30 a. m.: Patient suddenly became cyanosed and pulseless and ceased to breathe.

AUTOPSY AND REPORT BY DR. HARRISON S. MAITLAND.

**Head.**—In the cortex of the middle of the right frontal lobe is a small, cortical and sub-cortical abscess, measuring about 1.5 cm. in size and filled with thick, creamy pus containing a few small fragments of bone; the wall of the cavity is about 2 mm. in thickness.

Situated deeper in the brain substance, its outer edge encroaching on the small abscess already described, is another more recent and larger abscess, measuring about 2.5 cm. in diameter, and containing a slightly greenish pus; its walls are about 1 mm. in thickness. The lower and inner portion of this abscess cavity, about 1.5 cm. in size, leads directly into the anterior horn of the right lateral ventricle, the opening being soft and necrotic brain tissue.

The right ventricle is distended with greenish-yellowish pus, and there is an extensive exudate clinging to the choroid plexus; the ependyma lining the cavity contains numerous punctate hemorrhages, and the surrounding brain tissue is soft, œdematous, and in places distinctly necrotic. This same condition is found in the left lateral ventricle, although not to so marked an extent.

The third ventricle contains purulent exudate and fluid pus, and the interpeduncular cisterna is distended with purulent exudate. The fourth ventricle is dilated, as is also the iter, and contains a purulent exudate and free pus. This has leaked out through the foramina of Magendie and Luschka and infected the cisterna magna, which is filled with a plastic, purulent, yellowish exudate, which extends up over the inferior and lateral surfaces of the cerebellum and stops. The cisterna basilis and chiasmatis contain considerable plastic exudate, which apparently comes from extension from the cisterna magna, and possibly also from the close proximity of the infected third ventricle. The outer surfaces of the brain and the spinal cord and its meninges are practically free from exudate.

Extending from the wound of entrance in the middle of the frontal lobe passing across the frontal lobe on its superior surface, to the left and slightly backward, through the left frontal lobe on its superior surface, and stopping at the bone opening in the left parietal region, is a somewhat collapsed brain tract of the old bullet wound. The brain tissue is stained a gamboge yellow around this old tract and there are numerous piarachnoid adhesions over this area.

**Comments.**—Of course, a brain abscess should have been suspected, and an attempt made to drain it, possibly associated with subarachnoid irrigation. Subarachnoid irrigation, however, would of course have been useless without the removal of the cause.

In view of the excellent condition of the patient just prior to the second irrigation—with the rapid rise of blood pressure and the sudden termination, one is forced to the conclusion that death resulted from cerebral compression, probably induced by the irrigation. In addition to the over-looked abscess, the patient probably was over-treated both as to lumbar puncture and serum injections, and subarachnoid irrigation.

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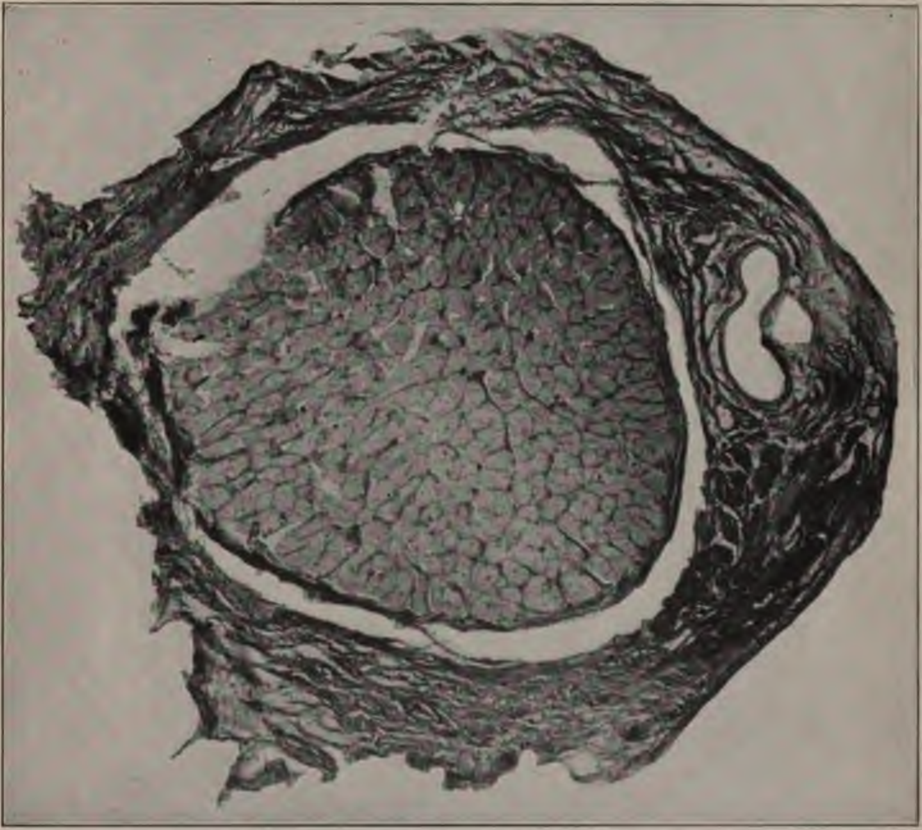

FIG. 33 (a).—Cross section of left optic nerve—showing perineural infiltration with thrombosis of small vessels and degeneration of the nerve fibers in a case of frontal lobe abscess which, after evacuation of the abscess, perforated the lateral ventricle and was followed by a closed empyema of the ventricle, causing blindness. The O.E. picture was that of strangulation of the vessels—"Stupeur Artérielle." (The shrinkage of the sections due to the hardening process in formalin is proportional in both right and left optic nerves; the atrophy due to pathological conditions was much greater in right optic nerve than in left.)

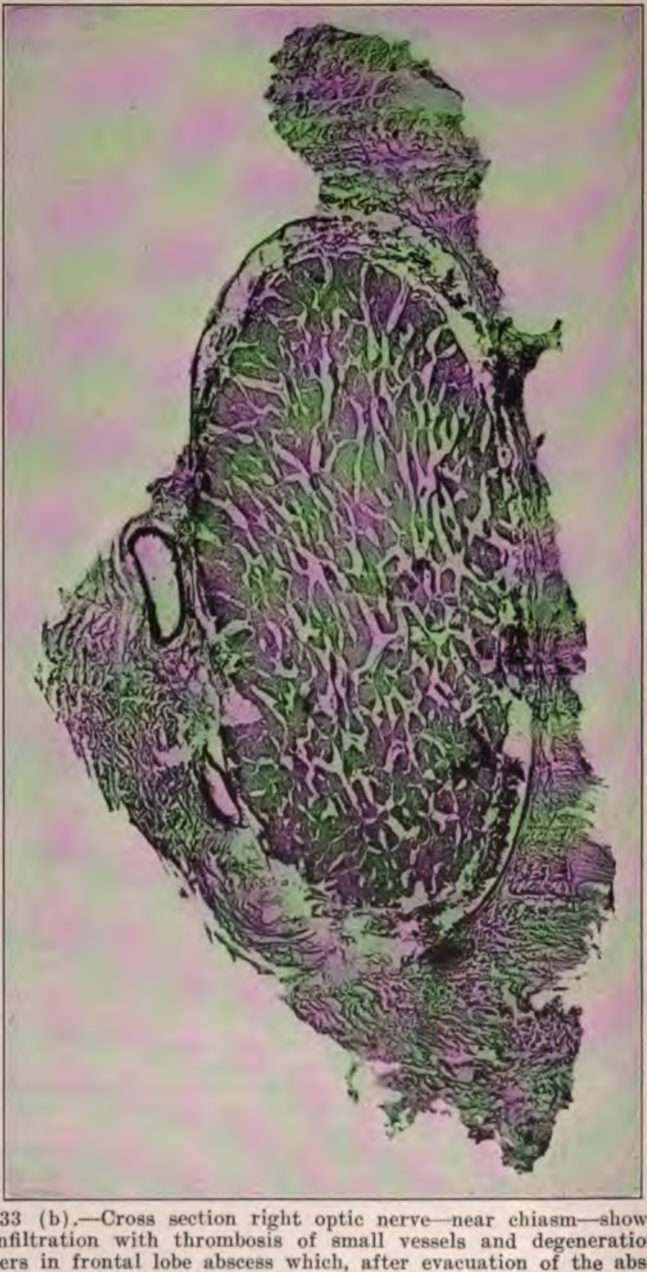

FIG. 33 (b).—Cross section right optic nerve—near chiasm—showing perineural infiltration with thrombosis of small vessels and degeneration of the nerve fibers in frontal lobe abscess which, after evacuation of the abscess, perforated the lateral ventricle and was followed by a closed empyema of the ventricle, causing blindness. The O.E. picture was that of strangulation of the vessels—"Stupeur Artérielle." (The shrinkage of the sections due to the hardening process in formalin is proportional in both right and left optic nerves; the atrophy due to pathological conditions was much greater in right optic nerve than in left.)

# PART III.

# SURGICAL DIAGNOSIS.

# CHAPTER X.

## DIAGNOSIS OF BRAIN ABSCESS IN GENERAL.

In the presence of a known focus of infection, either from the ear, nose, lungs, or other organ, with cerebral symptoms, brain abscess is to be differentiated from

(1) blood-stream infection,
(2) meningitis,
(3) vascular lesions,
(4) brain tumors.

The symptomology peculiar to brain abscess should be divided into

I—EVIDENCES OF CEREBRAL SUPPURATION.
II—SYMPTOMS OF CEREBRAL COMPRESSION.
III—LOCALIZING SYMPTOMS.

In the usual descriptions all the symptoms are intermingled more or less indiscriminately. This not only causes confusion, but often actually hinders an early diagnosis, for if the graver symptoms of cerebral compression—papillœdema, slow pulse, coma, etc.—are absent in the early stages of the abscess, to include them in the description minimizes the importance of the signs of cerebral suppuration which are always present and, while never so outspoken as those of cerebral compression, are capable of yielding a diagnosis at an early period if properly interpreted.

The literature is filled with records of cases where a diagnosis was not made "because of the absence of the, so-called, symptoms of brain abscess." Review of these cases will show that what in reality prevented the surgeon from making a diagnosis was not the absence of the symptoms of cerebral suppuration—for they were present—but the absence of those symptoms which point to an increase in the intracranial contents, the signs of cerebral compression.

Few cases of brain abscess present evidences of cerebral compression in the early stages; the majority of them not for some time, as the bulk of the brain is not increased; while a considerable number, especially those in which the frontal lobe is the site of the abscess, never cause compression. The importance of this fact is not generally appreciated.

In a doubtful case—and most cases are doubtful in the early stages—the surgeon should approach the subject with a broad viewpoint. In attempting a diagnosis he should adopt a definite mental policy. He should endeavor to distinguish between the manifestations of cerebral suppuration on the one hand and those of compression and localization on the other—though both of the latter may be absent.

In the course of his experience the author has adopted the following policy: (1) he asks himself (*a*) is cerebral suppuration possible? and (*b*) if possible, is it probable?

Should these questions be answered in the affirmative, he then tries to decide (2) is cerebral compression present? Then, and not until then, is (3) the localization of the suppuration, to be considered, when in the majority of cases the solution of the three former questions will have made apparent the probable site of the abscess.

(1-*a*) **Is Cerebral Suppuration Possible?** In a case that presents cerebral symptoms the absence of evidences of suppurative disease of the ear, nose, lungs, or heart at the time of examination removes the probability of a brain abscess. Although cases of otitic brain abscess are on record in which the abscess has remained quiescent for a long period of time during which the ear ceased to discharge, the author believes that a careful examination of the history of such cases would reveal that marked cerebral symptoms accompanied or immediately followed the aural suppuration.

On the other hand an accompanying focus of suppuration in the ear, nose, or lungs (bronchiectasis), renders possible a localized intradural suppuration. In such a case, although the cranial symptoms may be indefinite, the patient should immediately be admitted to a hospital for observation and routine examination.

(1-*b*) **Is Cerebral Suppuration Probable?** In the presence of cerebral symptoms with a known focus of suppuration of the ear or nose, intradural suppuration becomes not only possible, but probable. In such cases an early positive diagnosis is rendered possible by attention to a multitude of details to each of which the surgeon must give careful consideration. During this stage there generally is no one symptom which is pathognomonic. Too much significance should not be attached to the presence or—more especially—the absence of any one symptom. The surgeon must construct the picture by attention to minor evidences and after its construction he must consider it as a whole.

### I—EVIDENCES OF CEREBRAL SUPPURATION.

(*a*) **Initial Vague Chill.**—The "initial vague chill" indicates the beginning of an intradural suppurative process. It occurs when the infection invades the brain tissue. This initial vague chill generally is described by the patient as "not a chill, but a chilly feeling," and consequently the account of it is obtained only by a systematic inquiry into the patient's history. Careful questioning will establish not only the occurrence of the chill, but also the fact that from the time of its occurrence the health of the patient was not so good as previously to it. The eliciting of the exact date of the "initial vague chill" is of the greatest clinical importance, since from that can be computed the probable duration of the abscess, the degree of virulence of the suppuration, and the possible presence or absence of an encapsulating membrane, all of which will greatly influence the surgical procedure to be adopted.

(*b*) **Headache.**—This, in the author's opinion, is always present. It will vary from a dull headache to severe attacks in which the patient may become almost maniacal from pain. No other one symptom is of such uniform occurrence, or of greater assistance to a positive diagnosis. Its importance frequently is minimized by the patient; he "always has had headaches." Psychologically, there exists a disposition on the part of both patient and physician to disregard the headache because of this previous disposition. Questioning, however, will demonstrate that the headache is of somewhat different type from that previously complained of. Usually it is necessary for the surgeon to point out the difference to the patient.

In the presence of a known focus of suppuration in the ear or nose, very severe headaches preceded by a vague chill render an intradural suppuration extremely probable. In one of the writer's cases these were the only evidences demonstrable at the time of operation; and in numerous instances no other symptoms appeared for a long time, although the patients all along were suffering from encapsulated brain abscesses.

It is to be understood, however, that encapsulated, aseptic serous meningitis may cause this syndrome; but a localized meningitis frequently is the initial stage of an intrapiarachnoid abscess.

(*c*) **Vomiting.**—As with headache, the importance of vomiting as a symptom of brain abscess frequently is not appreciated, especially if the patient heretofore has had "vomiting spells" associated with headache. True projectile vomiting rarely occurs in the earlier stages of intracranial suppuration prior to the appearance of compression; but irregular vomiting without a definite cause is a frequent occurrence and should always be regarded as presumptive evidence of meningeal or cerebral suppuration. In one of the author's cases a boy, nine years of age, had for several weeks following a mastoid operation no symptoms of cerebral suppuration other than irregular headaches, two or three intermittent attacks of vomiting, and a slight peevishness of disposition, yet during this time there was a large collection of pus in the temporo-sphenoidal lobe. Examination of the recorded cases of frontal lobe abscess shows that in a large proportion of them headache and vomiting were the only symptoms present until shortly prior to death.

(*d*) **General Malaise.**—Loss of appetite, coated tongue, and dry skin always are present to a greater or less degree with cerebral suppuration. In cerebellar abscess the loss of flesh is one of the most characteristic symptoms. The combination of headache, vomiting, and general malaise, each or all out of proper proportion to the manifest local condition, preceded by a vague chill and a known focus of suppuration, associated with a high cell count in the cerebro-spinal fluid, calls for an exploration within the skull.

(*e*) **Disproportion.**—(*Value of, in diagnosis of cerebral suppuration*): A certain disproportion always exists between the commonly recognized symptoms of cerebral suppuration. This has its value in diagnosing between the symptoms that arise from the causative lesion—mastoiditis, or frontal sinusitis—and the symptoms of cerebral suppuration. The causative agents of a brain abscess—mastoiditis or a frontal sinusitis, themselves suppurative processes—produce the same general symptoms as intracranial suppuration, (chills, pain, headache, vomiting, temperature, general malaise and high cell count), and this complicates the problem. The symptoms originating from each, however, frequently can be distinguished by a study of the proportional relationship between the lesion and the severity of the symptoms.

In a determination of whether the symptoms presented arise from intracranial suppuration or from the mastoiditis, the surgeon must visualize the possible course of the different pathological processes. In the case of the boy referred to, he had a mastoid; an extradural abscess had been found at operation, but after its evacuation he became peevish and had irregular vomiting and headache. A child of nine years, recovering from a mastoid operation, might have attacks of vomiting from overeating. The disproportion was evident in this case, therefore, because children of that age rarely have associated vomiting and headache without an intracranial involvement. An adult, on the other hand, might be liable both to vomiting and to headache, but in the boy's case the repetition of such an association would signify a complication of the mastoiditis, or at least a pachymeningitis interna—itself a stage of intracranial suppuration—or a serous meningitis.

But, it may be asked, is not a pachymeningitis interna generally harmless? And are not pachymeningitis interna and serous meningitis protective processes, both self-limited? True. And do they not frequently disappear upon removal of the cause? True, also. Then why not reopen the mastoid and thoroughly clean it out? The answer lies in whether, in an individual case, a pachymeningitis interna or a serous meningitis could exist alone. In the case cited the diseased mastoid had been thoroughly exenterated, but in spite of this the peevishness, headache and vomiting subsequently appeared. Consequently, it could not be a pachymeningitis interna, because if that had been present at the time of the operation it would have been cured, and it had no reason to develop subsequently as the diseased bone had been thoroughly removed. The disease could not have followed this sequence. If the operation had been incomplete, or if a sinus thrombosis had existed, then a pachymeningitis might have been present.

A visualization of the possible intracranial course of the individual lesion will reveal any disproportion between the symptoms due to mastoiditis or to sinusitis alone. The following chart is offered as an aid to such visualization:

*Sequences in Adjacent Pathological Processes Entering the Brain.*

In the case of Mastoiditis, acute; there may be an associated
Extradural Abscess;
Pachymeningitis Interna;
Toxic localized leptomeningeal effusion—Serous Meningitis.

Mastoiditis, acute or chronic; may be followed by
(Extradural Abscess);
Brain Abscess from direct extension of suppurative tissue or retrograde thrombophlebitis.

Mastoiditis, acute or chronic; may have caused a
Labyrinthine or Perilabyrinthine involvement; from which may originate
Cerebellar Abscess from direct extension or retrograde thrombosis.

Mastoiditis, acute or chronic; may be followed by
Labyrinthine or Perilabyrinthine involvement; and at the same time cause a
Sinus Thrombosis; from which may originate
Brain Abscess secondary to sinus thrombosis.

**Mastoiditis;**
**Sinus Thrombosis;**
**Brain Abscess.**

**Frontal Sinusitis;**
**Pachymeningitis Externa (extradural abscess);**
**Meningeal Abscess.**

**Frontal Sinusitis;**
**Brain Abscess from direct extension of suppurative tissue.**

**Frontal Sinusitis;**
**Brain Abscess from retrograde thrombosis.**

### POSITIVE EVIDENCES OF CEREBRAL SUPPURATION.

(*a*) **Protective Meningitis.**—During the past few years, whenever infection of the ear or nose is present with cerebral symptoms, the demonstration of a protective meningitis by lumbar puncture has become one of the cornerstones of the author's diagnosis. A high cell count in the cerebro-spinal fluid from lumbar puncture is clinical evidence of intracranial involvement sufficient to warrant an intracranial exploration. The protective meningitis may be the evidence of a serous meningitis from the irritation of a neighboring infected focus, but if the adjacent suppuration in the ear or nose has been fully removed, the persistence of a protective meningitis is absolute evidence of intracranial suppuration.

In a high cell count the character of the cells is of the utmost importance. A high per cent. of polynuclears in the early stages of the disease indicates that the process is an active one, such as a serous meningitis from extradural irritation or from an irritation caused by an intracerebral abscess which approaches the surface. The cells, if largely mononuclear, show a subsiding process, for the function of mononuclear cells is to remove the debris from the intraleptomeningeal spaces. In meningococcus meningitis Neal[1] has shown that as the infection subsides the pus cells disappear and the percentage of mononuclears increases.

In Case XVII[2] the following report of the cerebro-spinal fluid examination was of material assistance in differentiating between a possible brain abscess and a cerebral vascular lesion: "Pressure slightly increased; transparency—clear, watery; no sediment after centrifugalization; globulin—moderate increase; cells—21 to the cmm., mostly polynuclears; Fehling's reduced. Bacteriological examination—sterile. Conclusion—secondary protective meningitis, probably from brain abscess."

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[1] Neal, Josephine B.: Meningeal Conditions Noted During the Epidemic of Influenza; *Department of Health of City of New York, Reprint*, Series 77, February, 1919, p. 4. "The mononuclear cells and the normal reduction of Fehling's solution are practically never present in a pyogenic meningitis."

[2] Case XVII, Miss M. D.; Chapter Five, Metastatic Abscess, p. 77.

The cell count may not be increased although an intracerebral abscess may be present at the time. In fact, the cell count is increased only when the meninges themselves are irritated, or during the stage of localization of the suppurative process. In intracerebral abscess from retrograde thrombophlebitis the cell count is not increased as long as the abscess does not approach the meninges. The writer's conception is that a high cell count in the cerebro-spinal fluid, with increase of globulin, occurs at an early stage in nearly all cases of brain abscess which originate by direct extension from the ear or nose.

For an understanding of the absence of a high cell count the course of the pathological process must be considered. During the course of localization of a meningeal process or a cerebral suppuration, the evidences of the protective meningeal reactions are exhibited in the general cerebro-spinal fluid for a few days only, as experimental evidence shows that after the production of an aseptic meningitis the cell count for the lumbar fluid is high only during the active stage, while the process in the cerebral meninges, although limited, may still be present. After the abscess is walled off from the meninges and the surrounding cerebral substance, the cerebro-spinal fluid obtained by lumbar puncture is nearly normal in constitution and the cell count, also, becomes normal. Later, when the abscess approaches the meninges, the cell count will again increase.

A high cell count, then, is important in diagnosis, but its absence should not militate against the probability of an intracerebral suppuration. In its absence the possible duration of the suppuration should be considered. In one of the author's cases,[3] on the eighteenth day of the disease, although an abscess was present in the cerebellum, the lumbar puncture fluid was clear and escaped rapidly by drops (consequently it was not under much pressure); reduced Fehling's, and contained only five leucocytes to the cubic centimetre. In bloodstream infections there is no protective meningitis and the cell count in the cerebro-spinal fluid remains normal.

(*b*) **Convulsion.**—In the author's experience a convulsion occurring in the presence of suppuration in the ear or nose is a positive sign of intracranial involvement. Although a few cases are recorded in which the removal of the focus of primary infection without the opening of the dura was followed by the disappearance of all symptoms, he is of the opinion that a localized serous meningitis

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[3] Case No. XXXI, W. J.: Chapter Twelve, p. 203.

—the first stage of an intrapiarachnoid abscess—existed at the time. In four instances in which the conservative course of removing only the adjacent foci of infection was adopted, all subsequently were found to be suffering from brain abscess, although the primary operation on the mastoid or nasal sinuses was followed by a period of improvement, an improvement which in one case lasted for several weeks. In the light of these experiences the author regards convulsions as positive indication of an intracranial involvement and their subsidence on the removal of the infected foci should not blind us to the fact that the intradural contents were affected. Association of convulsions with a known focus of infection calls for exploration within the dura.

In frontal lobe abscess, especially in children, convulsions are frequent. In one case to which the writer was called in consultation a child had two convulsions following orbital abscess. When first seen the child appeared perfectly normal. An intradural operation was refused. A surgeon subsequently operated on the ethmoid alone, disregarding the earlier convulsions; death followed in two days, with indications of a ruptured abscess.

Convulsions are of relatively infrequent occurrence in adjacent brain abscess except as a terminal manifestation of meningeal or ventricular rupture. It is to be remembered, however, that irritation of the temporo-sphenoidal lobe from brain tumors is much more apt to be associated with convulsions than is an irritation in any other portion of the brain. Especially with children it has been the author's experience that the first symptom of brain abscess was a sudden convulsion—in two cases occurring while the child was at play apparently in perfect health, although suffering from a chronic running ear.

In an examination of the recorded cases the author has been impressed with the comparatively frequent occurrence of convulsion in metastatic abscess. The explanation probably is that in metastatic abscess the cortex often is suddenly involved before a protective process has had time to guard the brain from the septic invasion. In adjacent intrapiarachnoid abscess such protection takes place before the cerebro-spinal fluid accumulation becomes purulent.

From observation the author is inclined to regard the occurrence of convulsions in brain abscess as suggestive either of (1) cortical involvement by an intracerebral abscess—convulsion being the expression of a more or less generalized cortical instability;[4] (2) abscess of metastatic origin and consequently not apt to be situated adjacent to the site of suppuration;[5] or (3) abscess of the temporo-sphenoidal lobe.

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[4] MacRobert, Russell G., and Feinier, Laurent: Cause of Epileptic Seizures in Tumors of the Temporo-sphenoidal Lobe; *Journal American Medical Association*, February 19th, 1921, p. 500.

(*c*) **Subnormal Temperature.**—During brain abscess the body temperature remains irregularly subnormal—a fact which is of the greatest diagnostic importance. No satisfactory explanation has been offered to account for the failure of a localized suppuration within the cerebral tissue to give the usual body reaction to a bacterial irritant—i. e., rise of temperature.

The theory that failure to maintain the normal body temperature is due to interference with the heat-regulating mechanism of the brain by cerebral suppuration—on the supposition that the brain and spinal cord possess two sets of cells which cause an increase or a loss of heat according as one or the other is stimulated,[6] should be abandoned, although importance still is to be attached to the effect of stimulation of the vaso-motor center. There is experimental evidence that the heat-regulating centers are situated in the basal segment of the mesencephalon. It has been found that removal of the cerebral hemispheres and the thalamus destroys the mechanism for regulating the body temperature.[7, 8, 9]

The daily normal temperature curve is due to fluctuation in tonus of the vegetative system, the center for the regulation of the tonus being situated, it is believed, in the hypothalamus. The secretions from the endocrine organs—the thyroid, pancreas, and adrenals—influence the tonus by stimulating the tonus center. Over-stimulation from over-action of the thyroid and other glands causes a moderate rise in the temperature curve, while diminished tonus—a fatigue phenomenon—is followed by a lowering of the normal daily temperature curve; both actions, stimulation and depression of the tonus, occur irrespective of the metabolic—digestive—processes.[10]

It has been established that the secretions of the brain contribute somewhat to the maintenance of a normal body temperature. Crile's[11] conception is that there is a normal secretion from the brain tissue itself, as well as from the pituitary and other glandular bodies, which contributes to the maintenance of the body temperature. The easily induced subnormal temperature of old age is a cerebral tissue manifestation associated with degenerative cerebral changes as shown by the cholin in the cerebro-spinal fluid. The subnormal temperature of hibernating animals is associated with an under-secretion of known glandular cells during hibernation.

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[5] Sautter, C. M.: *Journal American Medical Association*, February 5, 1921, Vol. 76, No. 6, p. 378. Also, *Laryngoscope*, December, 1920, p. 823. Reports a case of a boy of thirteen with a temporo-sphenoidal lobe abscess of otitic origin. Ten days after a simple mastoid operation convulsions intervened and the patient sank into deep coma. A decompressive operation was followed by improvement. Six days later convulsions returned. Evacuation of pus from area of frontal convolutions. Recovery.

[6] Adami and McCrae: Textbook of Pathology, 1914, p. 146.

[7] Rogers, Fred T.: Relation of the Cerebral Hemispheres to Arterial Blood Pressure and Body Temperature Regulation; *American Journal of Physiology*, 1919, Vol. 49, p. 271.

[8] Rogers, Fred T.: Studies on the Brain Stem—IV: On the Relation of the Cerebral Hemispheres and Thalamus to Arterial Blood Pressure; *American Journal of Physiology*, December, 1920, Vol. 54, No. 2.

[9] Rogers, Fred T., and Wheat, D. S.: Studies on the Brain Stem—V: Carbon Dioxide Excretions after the Destruction of the Optic Thalamus and the Reflex Functions of the Thalamus in Body Temperature Regulation; *American Journal of Physiology*, September, 1921, Vol. 57, No. 2.

[10] Mayer, A.: Pathologic Physiology of Human Body Temperature; *Deutsche Medizinische Wochenschrift*, 1919, Bd. 452, S. 1382.

In a general way hibernation resembles brain abscess in its symptoms. Hibernating animals have "subnormal temperature, tachycardia, slowed respiration, with diminished output of carbon dioxide, lowered blood pressure, relative peristaltic activity and marked insensibility to painful and emotional stimulus"—symptoms closely simulating cerebral suppuration.

### ADDITIONAL FACTORS IN SUBNORMAL TEMPERATURE—ABSENCE OF LYMPHATICS; AUTOLYSIS.

Cerebral suppuration being associated with an irregular subnormal temperature, the intermittent rises probably are due to a slight leptomeningeal space involvement. The author suggests that a factor is the absence in the brain of lymphatic channels which in the body tissues in general play such a large part in the conveyance and control of infection.

The delicate perivascular spaces surrounding the blood vessels empty into the subarachnoid spaces and are the channels whereby the dead tissue or other deleterious substances are removed from the brain. The phagocyte cells either absorb them or they are emptied into the blood vessels of the cerebral tissue, or are carried from the leptomeningeal spaces by the arachnoid villi. The high temperature in meningitis is due to the micro-organisms or toxins passing into the general blood stream, probably through the subarachnoid villi. Drainage into the large venous sinuses carries off the blood-stream infection in a considerable proportion of the cases of meningitis.

Thrombosis of the blood vessels in the area of the cerebral suppuration not only obliterates the vessels, but causes the perivascular spaces to become so filled with cells that they are no longer capable of functioning, and thus the remaining connection with the general circulation is removed; for as long as they remain within the cerebral substance they may remain inactive.

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[11] Crile, George W.: Origin and Nature of the Emotions; *W. B. Saunders Company*, Philadelphia, 1915, p. 138.

It is a clinical fact of diagnostic importance that suppuration within the cerebral tissue, at a distance from the meninges, not only fails to produce the well-recognized reaction of suppuration—an elevated temperature—but, on the contrary, frequently is accompanied by an irregularly subnormal temperature.

The author suggests that possibly the absence of fever in brain abscess is due to the fact that a brain abscess has little or no communication with the general circulation, being cut off from the vascular circulation by thrombosis of the vessels, and from the cerebro-spinal fluid circulation by obliteration of the perivascular spaces by exudates; while the irregular subnormal temperature in brain abscess may result from a disturbance of the heat-regulating mechanism of the brain. The toxins or micro-organisms which cannot reach the general circulation may depress the normal temperature either by depression of the tonus center or by direct action of the heat-regulating center, or possibly by interference with the normal secretions of the brain, as occurs in hibernation.

The resistance of the brain substance to autolysis may also play a part, for of all the soft tissues, the brain and the skin are the least affected by autolysis. Cholin, which is not highly toxic, is liberated by the autolysis of nerve tissues.

## II—SYMPTOMS OF CEREBRAL COMPRESSION.

Nothing has done more to delay diagnosis than the habit of regarding the manifestations of compression as direct symptoms of brain abscess. In many cases there is no increase in the brain bulk in the earlier stages, the pathological process being largely necrotic, the result of nutritional death from a retrograde thrombo-phlebitis. Later there may or may not be an increase of the intradural contents from the growth of the abscess, with an associated or reactive œdema and obstruction to the cerebro-spinal system circulation.

It will be of assistance if we do not regard the symptoms of compression as distinctly those of cerebral suppuration, but as the result of increase in the brain bulk which may or may not be present at the time of examination.

Only a limited degree of compression is possible in a brain abscess. The extreme cerebral compression so frequently associated with cerebral tumors never is present in suppurative intracranial lesions. Emphasis is laid upon the distinction. A neoplasm increases the brain bulk either by displacement of the cerebral tissue or by infiltration into it; an abscess grows by the destruction of cerebral tissue. The reactive forces of nature are paralyzed in the presence of suppuration by a very limited degree of the suppuration, and the nutrition of still unaffected parts is so interfered with that the suppuration rapidly extends and death soon follows. Therefore, the manifestations of extreme cerebral compression seen in cerebral tumors are never witnessed in brain abscess.

### SPECIFIC SYMPTOMS OF COMPRESSION.

In the presence of cerebral compression, all the cerebral functions are lowered. We have low temperature, slow pulse, and stupor, in contradistinction to the excitation of meningeal stimulation from suppuration, when there is high temperature, a rapid pulse, and cerebral excitement.

(*a*) **Slow Pulse.**—A continuous slow pulse is a distinct symptom of cerebral compression, but when present it generally is associated with other signs—such as papillœdema or coma—which enable a diagnosis. Of greater importance, however, in the early diagnosis of brain abscess is the appearance of periods of slow pulse followed by a rapid return to normal. In abscess of the brain a slow pulse may be recorded for a few hours—probably due to an acute œdema—only to disappear as the œdema subsides or, what is more important, as compensation is established.

(*b*) **Blood Pressure.**—The blood pressure uniformly should be taken at frequent intervals during the time the patient is under observation, chiefly because of its later value when surgical assistance will be necessary. With acute cerebral compression the blood pressure will rise, but in brain abscess, as a rule, compression develops slowly, in which case no variation in the systolic pressure will be recorded. It has taken the author many years to realize that hourly blood pressure readings, although of the greatest diagnostic assistance in acute compression with involvement of the medullary centers, are of little or no value in subacute or chronic compression, the mechanism of the two conditions being unlike.

Cerebral pressure is synchronous with the pressure of the veins within the skull. If the intracranial contents are suddenly increased the splanchnic system is suddenly called upon to re-establish the equilibrium between it and the increased intracranial contents. Consequently, the blood pressure rises to prevent the threatened medullary anemia and immediate death. In chronic compression compensation is established by factors within the cranium itself, and consequently, although the intracranial tension may be high, the blood pressure is not greatly, if at all, elevated. In one of the author's cases of acute temporo-sphenoidal lobe abscess in which signs of compression developed early and rapidly, the blood pressure rose from 130 to 170, while under ether 190 was recorded. Immediately upon evacuation of the abscess the blood pressure fell to 155, and on complete evacuation to 130.

(*c*) **Pulse Pressure.**—In acute cerebral compression not involving the medullary centers a rising pulse pressure is of diagnostic importance, as normally there is a close connection between the systolic, the diastolic, and the pulse pressures. A rising pulse pressure means acute cerebral compression away from the medullary center. Whether it is of any diagnostic assistance in acute abscess cases the author is unable to say; but in traumatic cerebral cases it has been of the greatest assistance to him.

(*d*) **Projectile Vomiting.**—This is of very infrequent occurrence in cerebral compression. It is, however, of frequent occurrence in cerebellar abscess, even without marked cerebral compression.

(*e*) **Ocular Paralysis.**—Ocular paralysis is of no localizing value whatever, the external rectus being the most frequently involved in any suppurative lesion. Its value as a sign of intradural suppuration is also greatly mitigated by the fact of its frequent occurrence in suppurative processes of the mastoid cells or of the apex of the petrous portion of the temporal, perhaps from an exudate at the base of the brain. The sixth nerve as it passes over the apex of the bone may be very susceptible at this point to involvement from an inflammatory exudate.

Third nerve paralysis, also, is of no localizing value—as demonstrated by Cushing. It is apt to be compressed between the area of transmitted intracranial pressure and the internal carotid artery. It is of much more value, however, than a sixth nerve paralysis as a sign of cerebral suppuration; but both are of great value as symptoms of transmitted intracranial pressure. In one of the author's cases there was an external paralysis of the opposite side from a temporo-sphenoidal lesion.[12]

(*f*) **Coma.**—This generally is the terminal stage of compression. If we await the development of coma before exploration, the mortality will be high, as cerebral compression favors the extension of cerebral suppuration. If we would have a lessened mortality from brain abscess, the abscess must be evacuated before compression has damaged the cerebral tissues, paralyzed the reactive mechanism of the brain, and so damaged the surrounding tissues as to make them a favorable soil for an extension of the abscess.

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[12] Case IX, G. W. Chapter Four, p. 38.

(*g*) **Papillœdema.**—Clinically it is seldom possible to differentiate between an optic neuritis and a papillœdema. The nerve-head manifestations of brain abscess usually are a combination of both. The causes giving rise to them are not thoroughly understood.

The generally accepted view of cerebral compression as the sole factor in their production is not consistent with clinical and experimental evidence. While general cerebral compression plays a part, other causes undoubtedly are operative. A better understanding of the exact pathological conditions producing papillœdema becomes necessary because of the widely accepted belief that it is solely of mechanical cerebral compressive origin—a view which frequently has occasioned the fallacious inference that an absence of papillœdema is clinical evidence of lack of increase in the intracranial pressure and has led to unnecessary delay in diagnosis. In considering the significance of the presence or absence of papillœdema the following factors must be recognized:

(1) *The Presence of Increased Intracranial Pressure;*
(2) *Interference with the Venous Return Circulation from the Cranial Cavity* (sufficient to cause nerve-head changes);
(3) *Location of the Lesion* (whether or not it interferes with the cerebro-spinal fluid circulatory system);
(4) *Alteration in the Composition of the Fluid* (contained within the piarachnoid prolongations of the cerebro-spinal fluid system around the optic nerve, into which the parenchymatous fluids of the optic papilla and nerve are discharged);
(5) *Induced "Stupeur Artérielle"* (due to an involvement of the periarterial sympathetic from the action of toxemia or trauma upon the central sympathetic system.)

### (1) THE INFLUENCE OF INCREASED INTRACRANIAL PRESSURE IN THE PRODUCTION OF PAPILLŒDEMA.

Cerebral compression is not always manifested by papillœdema. The intense form of papillœdema—choked disc—is, of course, a symptom of greatly increased pressure, but a mild or even a severe form of compression may entail no papillœdema. Choked disc occurs when the extracranial portion of the optic apparatus suddenly is filled with fluid or distended with exudates and inflammatory products, as in obliteration of the iter from pressure by a cerebellar tumor.[13]

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[13] Warrington, W. B.: Serous Meningitis; *Quarterly Journal of Medicine*, 1914, p. 105.

The etiological features of choked disc are (a) distension of the intravaginal sheath, (b) infiltration into the substance of the optic nerve, and (c) venous stasis from interference with the return venous circulation from the eyeball or from the cranium itself, such as follows from the ligation of an enlarged jugular, with extensive thrombosis of the intracranial sinuses—the cavernous and the superior longitudinal.

Distension of the intravaginal space is not always accompanied by choked disc. In several cases of fracture of the base of the skull involving the apex of the orbit, the author has observed an immediate blanching of the nerve head, and on subsequent post-mortem the optic sheath was found to be filled with blood. This explains the clinical observation that hemorrhage into the sheath from fracture may not occasion a papillœdema, but a primary optic nerve atrophy may follow within a few weeks.

A high degree of increase of the brain bulk may be tolerated without papillœdema when the position of the lesion causes no obstruction of the intracerebral portion of the cerebro-spinal fluid circulation. If papillœdema were occasioned by increase in the intracranial pressure alone, or even by direct pressure upon the optic sheath, nerve-head changes would be an early manifestation of abscess of the frontal lobe, because of the location of the abscess almost directly above the optic nerve and chiasm.

In frontal lobe abscess, however, papillœdema rarely occurs, although an abscess of large size may be present. It is this almost uniform absence of papillœdema in frontal lobe abscesses that has been one of the causes of failure to diagnose them. Examination of the recorded cases of frontal lobe abscess reveals the fact that although papillœdema is very infrequent before operation, it frequently develops after evacuation of the abscess, demonstrating that an increase in the intracranial pressure from abscess in front of the central portion of the cerebro-spinal fluid system is not associated with papillœdema and that the addition of another factor is necessary.

The author has observed numerous cases of cerebral injuries which during life gave distinct evidences of increase of intracranial pressure—confirmed at autopsy—but showed no nerve-head changes whatever.[14]

### (2) INTERFERENCE WITH VENOUS RETURN CIRCULATION FROM CRANIAL CAVITY IN THE PRODUCTION OF PAPILLŒDEMA.

Intense choked disc may develop from an acute obstruction of the return circulation from the cranial cavity and retina, but the papillœdema of brain abscess never is due to an obstruction of the vascular supply to the nerve head alone.

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[14] Eagleton, W. P.: Fracture of the Skull; *Archives of Surgery*, July, 1921, Vol. III, pp. 140-153.

A moderate increase of the intracranial contents such as occurs in brain abscess can not give rise to sufficient pressure to occasion papillœdema by strangulation of the vessels of the optic nerve and papilla, but when pressure is applied to an area where it obstructs the cerebro-spinal fluid system or interferes with the discharge of the intracellular juices of the nerve head into the subarachnoid prolongations of the nerve it will occasion a moderate papillœdema.

Increase in intracranial pressure, by a temporary obstruction of the circulation, may occasion the tortuous and dilated veins and redness of the disc so frequently seen in septic processes, but from this temporary congestion the vessels quickly recover.[15]

Congestion of the disc from increased intracranial pressure does not lead to papillœdema. Although frequently described as the first stage, it probably has but little to do with the development of a true papillœdema.[16]

For the development of a true nerve-head swelling it is necessary that to a venous obstruction there be added an increase in the cerebro-spinal fluid system circulation. The increase in the amount of cerebro-spinal fluid from the irritation of an inflamed sinus wall, and disturbance in the outflow from alteration in its composition, undoubtedly is the origin of the mild papillœdema often seen in sinus thrombosis.

The writer has seen a choked disc with hemorrhages and blindness immediately follow a ligation of the jugular for jugular bulb thrombosis and occasion an immediate complete venous stasis. The venous stasis occurred because the ligated jugular was the main vessel for the return of blood from the cranial cavity—the opposite jugular foramen being very small—or because a thrombosis occluded the superior longitudinal sinus.[17] The intense papillœdema probably originated in part from an associated acute internal hydrocephalus due to increase of the cerebro-spinal fluid from irritation and from interference with the discharge of venous blood from the cerebral veins through the veins of Galen, its discharge through the arachnoid villi being at the same time prevented by the thrombosis of the longitudinal sinus. The increased amount of cerebro-spinal fluid with diminished outflow resulted in an acute distension of the ventricles in addition to the venous stasis.[18, 19]

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[15] Martin and Crowe state that pressure on the internal jugular vein may cause increase in the venous dilatation at the fundus. This observation, if verified, should be of some diagnostic value. (Martin, H. H., and Crowe, S. J.: Lateral Sinus Disease—Case Report; *Laryngoscope*, December, 1920.)

[16] Pick, L.: Pseudo Neuritis Optica—Oedem der Papilla Nervi Optici—Neuritis Optica; *Zeitschrift für Augenheilkunde*, 1904, Bd. XI, S. 221.

[17] Eagleton, W. P.: Circulatory Disturbances Following Ligation of the Internal Jugular Vein in Sinus Thrombosis—with Report of Case; *Archives of Otology*, 1906, Vol. XXXV, No. 2, p. 91. Case XXIII, chap. VIII, p. 129.

In another case a mild papillœdema on the homolateral side followed an operation for a cervical adenitis. The papillœdema originated when an ascending retrograde thrombus, which developed in the jugular vein in the neck, entered the cranial cavity.

### (3) Influence of Location of Lesion in the Production of Papillœdema.

Papillœdema appears at once when the central portion of the cerebro-spinal fluid circulation is suddenly interfered with, causing an obstructive internal hydrocephalus. Abscess of the cerebellum also frequently occasions a mild papillœdema, chiefly because the pressure upon the iter between the third and fourth ventricles displaces the median line of the cerebellum and thus interferes with the cerebro-spinal fluid circulatory system. The intense choking of the disc so frequently seen in brain tumor, however, never occurs in cerebellar abscess.

Obstruction of the central cerebro-spinal fluid system is the cause of the mild papillœdema in many cases of temporo-sphenoidal lobe abscess,[20] while the almost uniform absence of papillœdema in frontal lobe abscess is, primarily, due to the fact that the central cerebro-spinal fluid system is not involved, papillœdema developing only when the abscess attains a large size or another factor is added.[21]

Very large lesions involving the cortex alone, large hemorrhages, or growths, frequently cause but little change discoverable by the ophthalmoscope. Direct pressure on the optic chiasm exerted from below, (as in hypophyseal tumors confined to the sella), and consequently not interfering with the central cerebro-spinal fluid circulatory system, do not cause papillœdema. When situated above the chiasm, however, as in suprasellar growths involving the floor of the third ventricle, bilateral papillœdema usually is present although without great venous stasis. The author's experience with tumors of the hypophysis causes him to regard the absence of papillœdema as an indication either that the growth is suprasellar or that, having been primarily sellar in location, it has broken through the dural envelope and invaded the general cerebral cavity.

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[18] Stols: Ueber die Unterbindung der Vena jugularis interna; *Correspondenzblatt für Schweizer Aerzte*, 1918, Bd. XLVIII, No. 13, p. 415.

[19] Ruttin, E.: Venenektasie in retra auriculären Operationsfeld und Stauungspapille nach Jugularis Unterbindung; *International Centralblatt für Ohrenheilkunde*, 1919-11, Bd. IX, S. 301.

[20] Case X, Chapter Four, p. 43; in which lumbar puncture performed during operation liberated but little fluid prior to evacuation, but the tapping of the abscess immediately was followed by free flow from the needle—an evidence of obstruction.

[21] Bollack, J.: Stase Papillaire et Dilation des Ventricules au Cours des Tumeurs Cérébrales; *Annales d'Oculistique*, 1919, Tome 156, pp. 538-571.

### (4) Influence of Alterations in Composition of Parenchymatous Fluid *(of the nerve head and the optic nerve, which is discharged into the prolongations of the cerebro-spinal fluid system surrounding the optic nerve).*

Papillœdema in brain abscess frequently originates from a blocking of the pathways by which the parenchymatous fluids from the nerve head and the optic nerve are discharged into the subarachnoid prolongations of the optic nerve, the blocking being caused by changes in the parenchymatous fluid itself, or in the cerebro-spinal fluid.

#### Intracellular Pathways of Optic Nerve and Retina.

"The optic nerve is not an outgrowth from the brain to the terminus as are the other cranial nerves, but a growth from the nervous elements of the retina *into* the brain." [22] "Histogenetically it is merely an association path of the central nervous system corresponding to the other association paths."

Primitively, the retina is a part of the cerebral cortex. If at an early stage the "eyeball is removed and the optic nerve cut, the geniculate, the pulvinar, and the anterior quadrigeminal bodies do not undergo complete development." [23] The trophic center, therefore, which controls the development of these deep-lying ganglionic parts is in the retina.

The intracellular fluids of the anterior part of the eye are discharged through the ligamentum pectinatum, the canal of Schlemm, and Fontana's spaces; those from the posterior part of the eye, however, the retina and the optic nerve—the cerebral portion of the eye—discharge into the optic prolongations of the leptomeningeal channels of the cerebro-spinal fluid circulatory system.[24, 25]

Verhoeff's cases apparently would furnish evidence that if an optic nerve tumor involves the subarachnoid space a papillodœma develops, while atrophy alone appears when the space is not attacked. (Verhoeff, F. H.: Primary Intraneural Tumors (Gliomas) of the Optic Nerve; *Transactions American Medical Association—Section on Ophthalmology,* Boston, June 8-10, 1921, pp. 87-127.

This system of intraneural, pericapillary, and perivascular pathways of the retina and optic nerve frequently but erroneously is spoken of as lymphatic,[26] although there probably does exist a communication at the apex of the orbit of this system with the true lymphatic system.

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[22] Gradle, H. S.: Development of Human Eye—Ocular Embryology; *American Encyclopedia Ophthalmology,* Vol. V, pp. 3862-3920.

[23] Ryder: Development of the Eye; *Norris and Oliver System of Disease of the Eye,* Vol. I, p. 60.

[24] Weed, Lewis H.: Dual Source of Cerebro-spinal Fluid—Studies on Cerebro-spinal Fluid; *Journal Medical Research,* September, 1914, Vol. XXXI, No. 1 (New Series, Vol. XXVI, No. 1), pp. 21-117.

[25] Wegeforth, Paul, and Weed, Lewis H.: Analogous Processes of Cerebral and Ocular Fluids—Studies on Cerebro-spinal Fluid; *Journal Medical Research,* September, 1914, Vol. XXXI, No. 1 (New Series, Vol. XXVI, No. 1), pp. 167-170.

Normal cerebro-spinal fluid does not coagulate, but alterations in its chemical properties—from irritation or inflammation—causes it to become coagulable to a greater or less degree. The author suggests that it is the filling of the intravaginal space with a pathological coagulable fluid,[27] or alteration of the perineural, pericapillary, or perivascular fluids—thus causing obstruction in the pathways—which is the additional factor generally operative in the production of papillœdema in abscess of the brain; much in the same way that interference—by inflammatory or hemorrhagic exudates—with the filtration of fluids from the anterior portion of the eyeball by blockage of the canal of Schlemm will precipitate an attack of acute glaucoma.[28]

The most intense choked disc with hemorrhage and venous tortuosities that the author has observed, having its origin from a brain abscess occurred in a very small temporo-sphenoidal lobe abscess, situated adjacent to the posterior horn of the lateral ventricle, and in which the ventricle fluid itself at operation coagulated. Prior to the operation the intensity of the choked disc occasioned doubt whether it could possibly be due to an abscess.

It is probably the cellular reaction to the toxic influence of an altered cerebro-spinal fluid which is the cause of the mild papillœdema seen in many localized suppurative processes—such as that associated with an involvement of the cells at the base of the petrous pyramid which is accompanied by "Gradenigo's syndrome," all of which symptoms disappear on evacuation of the adjacent mastoid area. The toxic reaction from the altered cerebro-spinal fluid has here a direct pathway into an optic nerve prolongation from the interpeduncular and chiasmal cisterna. This is also the explanation of the frequently seen unilateral mild papillœdema associated with otitic sinus thrombosis—thrombophlebitis.[29]

[26] Behr, C.: Zur Entstehung der Stauungspapille; *v. Graef's Archiv für Ophthalmologie*, 1920, Bd. 101, S. 165.

[27] Weed, Lewis H.: Dual Source of Cerebro-spinal Fluid; *Journal Medical Research*, September, 1914, Vol. XXXI, p. 103. (States "Intra-leptomeningeal channels of pathological coagulable fluid probably cause obstruction of the perineural, pericapillary, and perivascular system of the optic nerve.")

[28] Schiek believes from his histological examination of six cases that papillœdema arises from an obstruction to the "axial output from the eye"—a lymph stasis at the papillar end of the optic nerve, especially from over-filling of the intravaginal sheath with cerebro-spinal fluid. Schiek, F.: Die Genese der Stauungspapille; *Centralblatt für Innere Medizin*, 1910, Bd. XXXI, S. 484.

[29] Rados produced papillœdema experimentally by the cerebral inoculation of extracts from virulent tumors. Rados, A.: Histologische Veränderungen an der experimentalen Stauungspapille; *Archiv für Augenheilkunde*, 1915, Bd. LXXIX, S. 199.

Lewinsohn demonstrated by animal experiments that the injection of glycerine, oil, or liquid paraffine into the subdural space caused a delayed change in the papilla, which must have originated from obstruction by the altered fluid and not simply from

### (5) INDUCED "STUPEUR ARTÉRIELLE" IN THE PRODUCTION OF PAPILLŒDEMA.[30]

Arterial contraction from disturbance of the central sympathetic nervous system in the brain—as the result of trauma or toxemia—undoubtedly is a factor in the production of nerve-head changes in certain cases. The author has seen a mild papillœdema from an enormous frontal lobe abscess followed, after evacuation, by obliteration of the arteries and veins, with blindness due to an accumulation of pus in the lateral ventricle of the affected side, as shown at post-mortem. The basal cisterna was also enormously distended. (Figs. 33 a and b.)

Contraction of the vessels of the nerve head frequently is an early manifestation of fracture of the skull from—the author suggests—a disturbance of the fibres of the sympathetic nerves surrounding the arteries and veins. On the following day the nerve head appears normal unless a hemorrhage of the optic nerve sheath has occurred. In these cases the fracture generally passes through the apex of both orbits.

In one case, following a fracture which went through both orbital roofs, both discs were white, the arteries being obliterated. On the following day after the removal of bone the discs were normal. In spite of the normal discs, however, greatly increased intracranial pressure was present, as was shown during life by the large amount of fluid under great pressure in the lumbar punctures, and later at autopsy by whiteness of the cortex and obliteration of the sulci. Both optic nerves were tightly held and indented where they passed through the orbital foramina, as if they were compressed. Microscopical examination of the nerve, however, failed to reveal any pathological change.

In another case a white nerve head accompanied an extensive hemorrhage of the head of a new-born infant. The white disc persisted after the removal of the clot. At post-mortem a large intrapiarachnoid abscess was found, with an obstructive hydrocephalus from closure of the iter.

increase of intra-cranial pressure, as this would immediately have been manifest. Lewinsohn, G.: Experimenteller Beitrag zur Pathogenese der Stauungspapille; *Archiv für Ophthalmologie*, 1912. Vol. 81, p. 15.

Klauber, from clinical and histological examinations of wounds of the head, found papillœdema to be more frequent in the presence of infectious complications, due to an endocranial lymph stasis from the toxic irritation caused by necrotic brain tissue and extravasated blood. Klauber, E.: Klinische und histologische Betrachtungen über das Oedem des Schnorenkopfes bei Gehirnverletzten; *Klinische Monatsblätter für Augenheilkunde*, 1918, Bd. LX, S. 504.

[30] Leriche, René: Some Researches on the Peri-arterial Sympathetics; *Annals of Surgery*, October, 1921, Vol. LXXVI, No. 4, p. 387.

### DIAGNOSTIC DEDUCTIONS FROM PRESENCE OR ABSENCE OF NERVE-HEAD CHANGES IN BRAIN ABSCESS.

(1) A moderate grade of papillœdema is a frequent manifestation of cerebellar abscess. It originates from an obstruction in the central cerebro-spinal fluid circulatory system (a) by cerebral displacement at the iter, or (b) by the accumulation of a pathological fluid from an associated arachnoiditis in the basal cisterna;

(2) The same explanation of displacement accounts for the papillœdema frequently seen in occipital lobe abscess, the pressure being applied from above, consequently necessitating the suppurative process being well forward in the occipital lobe;

(3) The presence of a papillœdema in a temporo-sphenoidal lobe abscess depends (a) upon whether the abscess sufficiently dislocates the brain to cause obstruction in the cerebro-spinal fluid system by pressure, or (b) upon the toxic action of an altered cerebro-spinal fluid in the nerve head. (In the latter case it primarily should be regarded as a manifestation, not of compression, but of cerebral suppuration.)

(4) The usual absence of papillœdema in frontal lobe abscess is due to the fact that—in spite of the greatly increased intracranial pressure—none of the above factors are present;

(5) Although when present papillœdema is of great diagnostic assistance in a case of brain abscess, its absence in the presence of other signs of intracerebral suppuration should not occasion delay in intradural exploration;

(6) Intense choked disc with swelling of several dioptres of the papilla and multiple hemorrhages never occurs from brain abscess alone. Its presence, when brain abscess is present, signifies that another factor also must be active—such as an acute, sudden obstruction to the venous return from the eye from thrombosis of, or pressure upon, the cavernous sinuses;

(7) Obliteration of the arteries of the nerve head, with blindness out of proportion to the visible ophthalmic changes, when associated with brain abscess is suggestive of involvement of the central portion of the sympathetic system such as would occur from empyema of the lateral ventricles.

## CHAPTER XI.

# DIAGNOSIS OF ADJACENT TEMPORO-SPHENOIDAL LOBE ABSCESS.

### LOCALIZING SYMPTOMS.

If, from a study of a case from the point of view of the symptomology of intradural suppuration in general, it is decided that a brain abscess exists, the next step in the diagnosis involves the localization of the pathological process.

*Localizing Value of Primary Focus of Infection.*—The important thing to be determined in the localization of a brain abscess in the early stages is the original point of entrance of the infection, since the abscess will be found adjacent to this except in the case of abscesses of metastatic character. The chief point in localization, therefore, is to determine whether the infection has entered the brain from the ear; whether it involves the labyrinth; whether it has entered from the nose or accessory sinuses.

If the labyrinth is involved the abscess is apt to be situated in the cerebellum; if there are no labyrinthine symptoms the abscess probably is temporo-sphenoidal; if the suppuration exists in the frontal nasal sinus, the abscess is apt to be adjacent to the posterior wall of the frontal sinus. In the latter case the X-ray is an important aid in localization.

In a brain abscess following aural suppuration the elimination of the cerebellum as the site of infection is of the greatest importance in localization; for, in adjacent abscess from the ear the abscess, if not metastatic, will be situated either in the temporo-sphenoidal lobe or in the cerebellum. If, in the absence of a spontaneous nystagmus, cold caloric in either ear in the upright position gives a normal nystagmus and normal vertigo, the cerebellum can probably be excluded. The pointing deviations are so uncertain that induced pointing variations may be largely disregarded.[1]

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[1] Barany, Robert: Temporary Depression of the Function of the Cerebellar Cortex after the Method of Trendelenburg, evidenced by the pointing test; *Journal of Laryngology, Rhinology and Otology,* August, 1911, Vol. 26, p. 444.

*Pathognomonic Symptoms in Temporo-sphenoidal Lobe Abscess.* —The temporo-sphenoidal lobe is a silent area of the brain. Even when it is the seat of a large collection of pus no localizing symptoms may be present. Two pathognomonic symptoms, however, frequently are present and outspoken—*i.e.,* aphasia (naming), and (an easily overlooked symptom) hemianopsia.

(*a*) **Aphasia.**—One of the author's cases suddenly manifested a sensory (naming) aphasia which lasted for a few hours and then entirely disappeared. In reviewing this case he is inclined to believe that in many of the adjacent temporo-sphenoidal abscesses which later develop aphasia, an incomplete aphasia is present off and on for several days prior to its becoming sufficiently outspoken to command the attention of the patient or the physician.

In a right-handed man with an abscess in the left hemisphere the presence of a naming aphasia is pathognomonic. The aphasia in the early stages is apt to be transient, appearing suddenly and as suddenly disappearing. At the time of the examination the patient may talk very well, but his family will state that on a certain date he was "out of his head." In one of the cases, careful questioning showed that the patient was unable to name an object which he wanted; after repeated attempts to name it, he got out of bed and got it for himself; but in a few hours the aphasia had disappeared entirely.

Personal experience has taught the writer not to be deceived by an apparent improvement. In one case a young man, following two operations on the jugular bulb, suddenly became aphasic. When seen on the following evening his speech was perfect, but from the description of his aphasia by his physician, the author felt warranted in advocating a temporo-sphenoidal exploration. On account of the previous operation and the—at the time—excellent physical condition of the patient, his family refused. A few weeks later he presented himself to all appearances in perfect health. Ophthalmoscopic examination revealed a slight papillœdema, however. The following summer he suddenly became comatose and died within a few hours from rupture of the abscess.

The reports of frontal lobe abscess contain several cases with a similar history. The transient nature of the aphasia undoubtedly comes from involvement by œdema of the associated tract; later the œdema is sufficiently abated to allow of complete restoration of function.

A "naming aphasia" is seldom complete until signs of compression develop. In the presence of suppuration within the ear, preceded by a vague chill, and associated with headache, exploration of the temporo-sphenoidal lobe is demanded. Even when complete, the aphasia disappears entirely after operation, so that the tract can not have been destroyed. The "naming" character of the aphasia, however, although incomplete, usually can be demonstrated if properly investigated. Frequently it can be elicited only by careful questioning.

When incomplete this type is rather definite. The patient, when he names an object correctly, realizing its correctness, will repeat the same name for the next object; but while he may or may not know that this is the wrong name, he invariably can use or properly describe the use of the object.[2]

(*b*) **Word Deafness.**—The labyrinth contains, developmentally, the end organ of one of the most primitive of the special senses and one of the latest of all—the vestibular, and hearing apparatus. If we except speech, audition is the most highly specialized of all our special senses. In spite of the general conception that the eye is the most highly specialized sense, hearing as we understand it, undoubtedly is. It is the only one of the special senses below speech which has even a tendency to be localized in one cerebral hemisphere. Hearing is a specialization of a special sense—that of sensation, touch—and it is out of the specialization of hearing that speech has been acquired by man.

While experimental and histological evidence would place the cortical center for hearing bilaterally in the transverse temporal gyri of Heschl and in two-fifths of the temporal convolutions,[3] a total deafness of the central organ is of rare occurrence for the reason that the hearing centers are bilateral—situated in both temporo-sphenoidal lobes—and consequently total central deafness follows only upon the destruction of both lobes.

The transverse temporal gyri, probably of both sides but certainly of the left side, contain the primary centers for the reception and analysis of auditory stimuli; they represent the uppermost link in the chain of auditory neurons.[4] Interference with the functioning of the cortical center for hearing such as would be caused by the pressure of a temporo-sphenoidal lobe abscess may give rise to a complex of symptoms which are of great diagnostic value. Of these, word deafness—a form of aphasia—is the most important. In general, aphasia is the inability to call a word, or the use of inappropriate words. Word deafness, as defined by Laiguel-Levastine,[5] is rather a "syndrome characterized by the absence of adaptation of the words to the ideas and of the ideas to the words."

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[2] Gordon, Alfred: A Contribution to the Study of Aphasia; *New York Medical Journal*, January 4, 1913.

[3] Kennedy, Foster: Symptomology of Temporo-sphenoidal Tumors; *Archives of Internal Medicine*, September, 1911, Vol. 8, pp. 317-350.

[4] Campbell: Histological Studies on the Localization of Cerebral Functions; 1905.

In one of the author's cases [6, 7] word deafness was a manifestation of a large, left-side, intrapiarachnoid temporo-sphenoidal lobe abscess. The patient, although possessing hearing in both ears, and affected by a Wernicke "naming aphasia" which allowed him to use numbers but not proper names, had a complete word deafness. He was unable to call, repeat, or understand any proper names, but recognized and appreciated objects by sight alone. When he desired anything —lemonade, for instance—he uniformly would say, "Help me—help me——;" then, realizing his inability to make his want known by words, would add, "Show me." When shown the right object—the lemon, perhaps—he instantly would recognize it as the thing desired and assent by saying "Yes."

He recognized his physician and friends but could not call their names nor even understand the names when they were spoken. Commands given him with nouns in the sentences he did not understand, but commands without nouns were understood, although imperfectly. When told to lie down he did so, asking, "Is this what you mean?" He was conscious of and annoyed by a paresis of his right arm and described it by saying, "All in."

Following the evacuation of his abscess the word deafness immediately disappeared, but the "naming aphasia" continued. He then could understand a name and could repeat it. When shown a knife, for instance, he could not say what it was, but when asked if it was a knife he not only recognized it but understood the name of it. He was imperfectly able to read printed numbers and letters, but apparently did not completely comprehend the command to read, as he generally tried to copy them instead of reading. He often inverted the letters and figures in writing them, but as he was writing with his left hand, the right still being paralyzed, this may account for it.

In the writing tests he made the figures 2 and 6 upside down; a 7 he wrote 9; and 8 he also wrote 9, and a 9 was written 10. For 24 he drew an indeterminate scrawl. The figure 2 he read two, but the 10 he called five. The letter W he read aright, but the letter P he was unable to read.

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[5] Laiguel-Levastine: Aphasie et Apraxie; *Le Bulletin Médical,* 1920, Vol. XXXIV, p. 277.

[6] Case VII. J. C. C.: Chapter Four, p. 35.

[7] Note: Cases of brain abscess with word deafness have been reported by Brown, W. G. Mastoid Disease with Cholesteatoma Complicated by a Cerebral Abscess; *Medical Journal of Australia,* 1920, I, p. 212; and Wiener, Alfred: A Case of Brain Abscess Following Traumatism and Acute Mastoiditis—Operation—Recovery; *Archives of Otology,* 1906, Vol. XXXV, No. 4.

(*c*) **Hemianopsia—Transient or Fixed.**—Hemianopsia is one of the most important localizing signs of temporo-sphenoidal lobe abscess. It is due to an involvement of the association fibers running from the cortical optical center in the cuneus to the geniculate bodies—the cuneo-pulvinar tract (Meyer's Tract)—which passed through the temporo-sphenoidal lobe. Cushing[8] has demonstrated that in temporo-sphenoidal lobe tumors this tract is caught between the tumor and the distended ventricle. This undoubtedly also is the explanation in temporo-sphenoidal lobe abscess cases.

In a brain abscess as well as in a brain tumor case the author has seen a complete hemianopsia for colors which disappeared entirely on the following day. In his experience the hemianopsia has been latent in all abscess cases and he believes that a transient hemianopsia is of much more frequent occurrence in temporo-sphenoidal lobe abscess than generally is appreciated, since it is not an outspoken symptom and not recognized by the patient himself, even when complete for form as well as for color. Consequently, if the surgeon is to avail himself of a localizing hemianopsia in making a diagnosis, it is necessary to examine the visual fields repeatedly both for form and for colors. (Fig. 34.)

Hemianopsia persists long after the subsidence of suppurative symptoms, and in some cases even after the abscess has been cured. In a re-examination of two of the author's healed cases of temporo-sphenoidal lobe abscess[9] hemianopsic sections of the field were still found wanting after an interval of three and five years. (Figs. 35 and 36.)

### CASE NO. XXVII.

E. L.: Hemianopsia a Deciding Factor in Exploring Temporo-sphenoidal Lobe. Boy.

In the case referred to on page 177 the abscess was of many weeks' duration, during which time the mastoid wound and drum membrane had entirely healed, the hearing returned to normal, and no nerve-head changes were present. The only suspicious symptoms during these weeks were irregular vomiting and a mild form of headache—the former having been attributed to errors in diet and the latter to eye-strain from a refractive error. Five weeks after the prescribing of the glasses the headache became suddenly severe, the vomiting frequent, an intense choked disc developed and the vision was reduced.

On examination a diagnosis of a probable brain abscess was made, based upon (1) the presence of a previous aural suppuration; (2) irregular vomiting and headache; localization of the pus in the temporo-sphenoidal lobe was suspected because repeated examinations of the field for form and colors had on one occasion—two days previously—shown a distinct tendency to (3) a hemianop-

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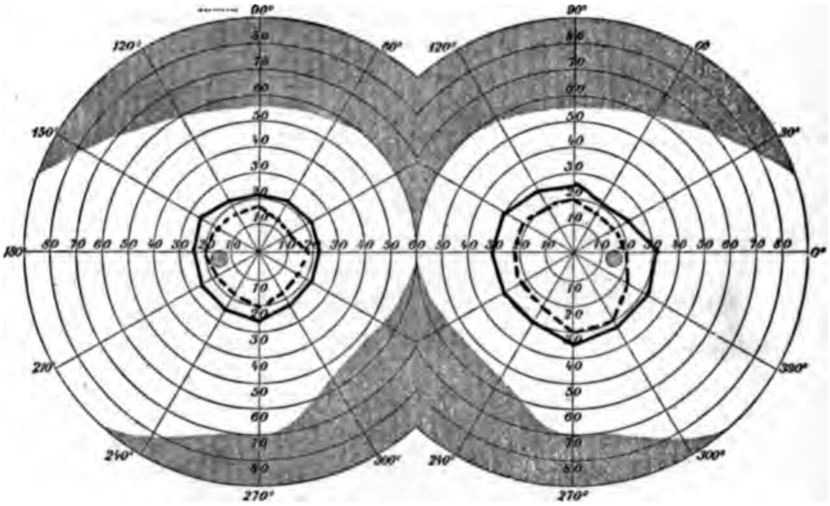
[8] Cushing, Harvey: *American Neurological Association*, Atlantic City, June, 1921.

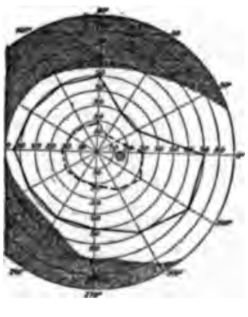
[9] Case X, C. H., Chapter Four, p. 43; and Case XVII, M. D., Chapter Five.



**Left Eye** **Right Eye**

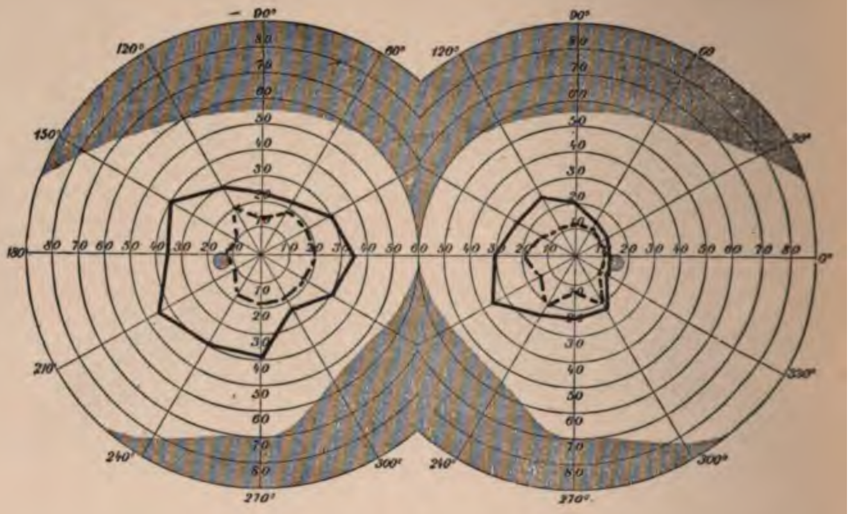
(Solid line ——— shows form field, dotted line ······· color field.)

FIG. 34.—Bilateral suggestive hemianoptic indentation of color fields in a case of left adjacent temporo-sphenoidal lobe abscess. (Case XXVII, E. L., Chapter Eleven, p. 180.)



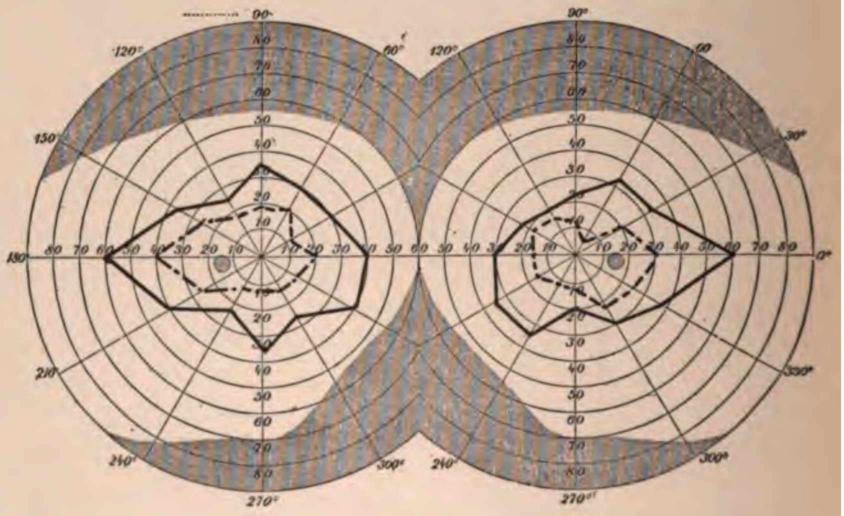
(Solid line ——— shows form field, dotted line ······· color field.)

FIG. 35.—Persistent hemianoptic indentation of field of right eye of patient three years after operation for a left-sided adjacent temporo-sphenoidal lobe abscess. (Case X, C. H., Chapter Four, p. 43.)



Left Eye Right Eye

(Solid line ——— shows form field, dotted line - - - - - - - color field.)



Left Eye Right Eye

(Solid line ——— shows form field, dotted line - - - - - - - color field.)

Fig. 37, Upper Figure.—Transient suggestive hemianoptic field in case of left adjacent temporo-sphenoidal lobe abscess.

Lower Figure—Two days later; disappearance of hemianopsia for form, but indentation for colors.

tic contraction in the eye of the affected side and a corresponding indentation in the opposite eye (Fig. 37); while the patient gave (4) a history of having a few days before had difficulty in recalling several names, among them the word "scapular"—a familiar word for a Roman Catholic who had worn one for several years.

The abscess, which was of very small size with a very firm capsule, was located near the posterior horn of the lateral ventricle. After the evacuation of about two drachms of thick pus in a misguided effort at complete evacuation, the searcher was forced into the adjacent lateral ventricle. The cerebro-spinal fluid discharged was so viscid that it formed a cast within the searcher. As the ventricle was not dilated—as shown by both ventricular and lumbar puncture—it undoubtedly was the change in the character of the fluid itself which occasioned the intense choked disc.

(*d*) **Facial Paralysis of Opposite Side.**—Generally the first manifestation of involvement of the motor area of the brain by an adjacent temporo-sphenoidal lobe abscess is a paresis of certain of the muscles of the opposite side of the face. This facial paresis is of the cortical, not of the peripheral type. It involves the lower portion of the face and is elicited only by spontaneous emotional movements, such as laughing, when the lower portion of the face appears flattened, while the eye, which the patient can close on command, does not close reflexly by volition so frequently nor so completely as the other.

Facial paresis is of the greatest importance in diagnosis. It long precedes weakness of the arm and the leg. In the presence of suppuration in the ear, with a history of vague chill and headache, a cortical paralysis of the face of the opposite side to the aural suppuration makes a diagnosis positive.

(*e*) **Paralysis of Contralateral Arm.**—A weakness—paresis or paralysis—of the arm, which comes and goes, is a frequent localizing manifestation of brain abscess.

(*f*) **Contralateral Hemiplegia.**—Complete hemiplegia in temporo-sphenoidal lobe abscess is a very rare condition. When it occurs it can be differentiated from a vascular lesion by the progressive nature of the paralysis, the face first becoming involved, then the arm, and last of all the leg, the involvement of the groups being separated by appreciable intervals. This is in contradistinction to vascular disease in which the onset is sudden and involves face, arm and leg simultaneously.[10]

In the presence of a complete contralateral hemiplegia which has appeared suddenly, the probability is that the surgeon is not dealing with an adjacent temporo-sphenoidal lobe abscess, since an adjacent abscess by reason of its location away from the internal capsule cannot cause a complete hemiplegia unless it is of enormous size, but with a vascular lesion or an abscess of metastatic origin. A metastatic abscess may cause a complete hemiplegia either (1) of internal capsular origin—its causative retrograde thrombophlebitis involving the vascular supply of the internal capsule—or (2) of cortical origin, the infecting nidus having been confined to the motor area by way of a secondary thrombosis of a dural or pial vessel, the intercurrent intradural or extradural abscess being situated over the motor area at least three inches above the ear.

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[10] Case XVII, M. D., Chapter Five, p. 77.

CASE NO. XXVIII.

P. Q.: Case of *Intercurrent Superficial Brain Necrosis, Situated Three Inches Above the Ear, Over the Motor Area of the Cerebral Cortex* (with Necrosis of Dura and Extradural Abscess) to which Point the Infection Was Carried from the Original Focus in the Middle Ear by an Intercurrent Thrombosis of a Dural Vein.

History: The man was brought to the hospital complaining of intense pain in his left ear; dizziness, vertigo, noises in his ear. He had had bilateral running ear in both ears for many years, but during the past year the discharge had stopped in his right ear; it had not stopped in his left. An examination revealed a dead labyrinth. The headaches and dizziness gradually disappeared and he was discharged from hospital. He was admitted the same day, with high temperature. It was now found he had a high cell count of his cerebrospinal fluid; it was under great pressure, and cloudy. He had a leucocytosis of 13,000. He was rigid and had the symptoms of meningitis. However, to the surprise of everybody, this cleared up and the patient was again discharged.

Four months later he was again admitted to the hospital. He now had a paralysis of the right arm. He had a slight facial paralysis of the right side. He had no "naming aphasia," but apparently had some difficulty in talking. He had two convulsions the night of admission and had had two or three the day before. He was apparently deaf in the left ear. He was able to walk and did not have a Romberg.

From the history of the case it seemed certain that the man had an encapsulated brain abscess of the temporo-sphenoidal lobe, though it was hard to account for the lack of "naming aphasia," and "how a right-handed man could have a right-handed paralysis of the arm and face without a 'naming aphasia,' if the abscess had extended upward from the tegmen in the ordinary way." It was also certain that the abscess was near the surface, because of the convulsions. For the same reason—because of the convulsions—it was judged to be a metastatic abscess, the author not having seen convulsions in an adult except in a metastatic abscess.

**Operation.**—A large osteoplastic flap was turned up in the usual way, over the middle fossa. On drilling the last hole—that is, the anterior hole, well up toward the vertex—very profuse bleeding occurred. On passing the searcher from this to the next hole there was a free discharge of pus. Another hole was drilled between number one and two—on a straight line—because of the adherence of the dura. About two drachms or half an ounce of pus was discharged from the two holes. The abscess was found to be extradural with necrosis of dura and superficial brain necrosis. A large dura vein, which was thrombosed, ran directly upward in the anterior portion of the exposed area. At the upper end of it, it formed the extra-dural abscess, which had been completely evacuated. An incision was made in the dura. Along its whole course the large thrombosed vein was firmly adherent to the brain underneath as well as to the dura. The brain was searched in the upper portion to a depth of about six centimeters, but nothing was found. The entire bone flap was sacrificed; tight closure and drainage from the exposed dura—the area of exposed brain—and another drain above.

Comment.—The case is remarkable being a metastatic abscess secondary to a thrombosis of a dural vein that originated over the whole of the arm area of the same side. A lumbar puncture done at the time of the operation revealed very little fluid. After the operation the paralysis of the arm gradually disappeared; temperature fell to normal; and the patient recovered.

### SYMPTOMS OF ASSISTANCE IN DIAGNOSING TEMPORO-SPHENOIDAL LOBE ABSCESS.

(*a*) **Pain in Teeth** on the side of the affected hemisphere, together with symptom of pain behind the eye must be due to involvement of the Gasserian ganglion at the apex of the petrous pyramid. In one of the author's cases of temporo-sphenoidal lobe abscess which followed a chronic suppuration of the petrous pyramid,[11] the pain in the teeth had been so severe that a dentist repeatedly had been consulted. On the development of the brain abscess the cerebral symptoms were so outstanding as to overshadow the pain in the teeth, although for several weeks that had been the most prominent symptom. As the post-mortem showed a very extensive involvement of the cellular structure of the petrous labyrinth, without destruction of the auditory apparatus, the author is of the opinion that the pain in the teeth was due to the bone lesion and not directly to the abscess.[12]

(*b*) **Pain behind the Eye.**—In two cases of temporo-sphenoidal lobe abscess in which drainage tubes had been inserted, irrigation occasioned pain behind the eye in the affected hemisphere whenever the cavity was distended by the fluid, the pain disappearing immediately upon the release of the fluid. The pain behind the eye was produced by each distension and could be uniformly produced as long as the drainage tube remained in place.[13]

The author has no evidence to warrant including it among the symptoms of temporo-sphenoidal abscess, but he is persuaded that pain behind the eye of the affected side should be considered as a localizing symptom of more or less value, depending upon the individual case. The pain behind the eye probably is due to irritation of the ophthalmic branch of the trifacial in the middle fossa, and not to irritation of the Gasserian ganglion.

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[11] Case VIII, A. M. M., Chapter Four, p. 37.

[12] Cadwalader, Williams B.: Significance of Facial Pain in Determining the Location of Intracranial Tumor; *Archives of Neurology and Psychiatry*, August, 1920, Vol. IV, pp. 182-184.

[13] Case XIII, Mrs. H., Chapter Four, p. 68.

Pain behind the eye of the homolateral side should not be confused with the pain behind the eyes of both sides which, the author has come to believe, is of considerable diagnostic significance; the bi-lateral, post-ocular pain being caused by a distension of the lateral ventricle from an obstructive internal hydrocephalus such as is seen in cerebellar tumors with obstruction of the iter. The distended ventricles cause irritation of the nervous distribution of the fifth to the dura, and a general headache follows; or, pressure downwards on the sensory root of the fifth as it enters the middle fossa from the posterior, irritates the first branch of the fifth.

(*c*) **Convulsions—Localizing Value.**[14]—While epileptic seizures are of much more frequent occurrence in *tumors* of the temporo-sphenoidal lobe than in tumors in any other location in the brain —noted in about fifty per cent. of all the recorded cases—they are very infrequent in *abscess* of the temporo-sphenoidal lobe. McRobert and Feinier [15] have shown that the cause of convulsions in temporo-sphenoidal lobe tumors probably is pressure involvement of the vascular system of the middle central area. This probably also explains the convulsions in Case XIV, Chapter Five, p. 70, in which convulsion was due to something other than pressure, as the patient had two convulsions after the evacuation of the abscess at operation.

(*d*) **Dreamy State.**—This occurs in a small proportion of cases and often is overlooked entirely or mistaken for the semi-somnolent condition which is common in cerebral compression. There is, however, a distinct difference between the two conditions.

In one of the author's cases there were dreamy state attacks from temporo-sphenoidal lobe tumor. The patient was admitted to the hospital in a semi-stuporous condition, accompanied by the statement that:

"About three or four weeks previously he complained of severe headache and told his friends he was losing his mind. This was followed by a convulsion, since which time he has been unable to do any work. During the past week he has been in a more or less semi-conscious condition but could be aroused."

The patient being unable to make a connected statement, the following account was obtained: "Had not noticed anything wrong until one month previous, when the patient's tone of voice changed and he 'said some things which seemed unnatural.' Later he would stop in the midst of a task and stare into space. About three weeks before his admission to hospital he had a fit. After that he stated frequently that he was losing his mind. For the past two weeks had been ill, sometimes complaining of pain in left side of head, frontal and parietal regions. Had several attacks of vomiting, not projectile.

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[14] See p. 161, Convulsion, a Positive Evidence of Cerebral Suppuration.

[15] McRobert, Russell G., and Feinier, Laurent: The Cause of Epileptic Seizures in Tumors of the Temporo-sphenoidal Lobe; *Journal American Medical Association*, February 19, 1921, Vol. 76, No. 8, p. 500. (In forty-one recorded cases had *grand mal* attacks, and in five minor cases lesser types of convulsions.)

Lately refused to have anything to do with his friends—said he thought they had something against him."

**Post-Mortem.**—Cortex normal; in left temporo-sphenoidal lobe two large areas about two inches long and one inch wide, of gliomatous infiltration, in the upper one of which were a large number of hemorrhages with secondary degeneration.

**(*e*) Psychic Manifestations of Toxic Delirium.**[16]—In one of the author's cases [17] a toxic delirium was the most outspoken symptom. The patient, a single woman past the menopause, having just previous to her attack been impressed by accounts of the white slave trade, was obsessed by this during the whole of her illness until after the evacuation of the abscess.

The author has knowledge of another case in which the toxic insanity caused the commitment of the patient to an insane asylum where she remained for several months. Although the attendant physicians were aware of her suffering from a discharging ear, no importance was attached to it until the post-mortem revealed a temporo-sphenoidal lobe abscess.

**(*f*) Uncinate Symptoms.**—There are no cases on record of uncinate symptoms in temporo-sphenoidal lobe, but the author believes this is due to a combination of vague mentality on the part of the patient and carelessness in the investigation of the history. The anatomical site of the temporo-sphenoidal lobe must so frequently involve the anterior poles of the lobe as to produce illusions of the sense of taste and of smell.

### CASE NO. XXIX.

M. C.: Case of Brain Abscess Illustrating Transient Nature of Aphasia and Paralysis, with Apparent Complete Recovery without Operation; Sudden Extension of Metastatic Abscess Five Months Later; Death. Male, 18 years of age.

**History.**—April. Had had discharging *right* ear for 17 years, which caused no other symptoms until one month previous, when he complained of considerable headache and had fever and several chills. He was seen by an ear specialist, who advised mastoid operation. Parents refused operation and family physician treated patient for a month for malaria because of chills and fever, although three blood examinations were negative for plasmodia. Operation; diseased mastoid, pre-sinus abscess, and apparently thrombosed sinus. The following day internal jugular resected and removed broken down clot from the sinus.

After operation patient had two chills and persistent headache. Two days after operation complained that he saw double—had to close his eyes in order to see well. (Undoubtedly had paralysis of internal rectus of left eye at this time.) This gradually disappeared. Temperature returned nearly to

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[16] Henderson, D. R., Muirhead, W., and Fraser, J. S.: A Case of Toxic Exhaustive Insanity Associated with Chronic Suppurative Otitis Media, Labyrinthitis, and Extradural Abscess; *Review of Neurology and Psychiatry*, Edinburgh, 1913, Vol. 17, pp. 565-576.

[17] Case XVII, Miss M. D., Chapter Five, p. 77.

The X-ray showed an area, semi-spherical in shape, of a shadow in the temporo-sphenoidal lobe. The shadow was due to the presence of gas in the upper part of the abscess, the result of micro-organic growth. (See Fig. No. 38.)

The X-ray was made when the patient was in a recumbent position. Post-mortem revealed the presence of anærobic micro-organisms which produced bubbles of air and fluid, changing its shape with the change in position of the patient, the straight posterior line showing the level of the fluid. In such a condition a positive diagnosis of brain abscess was possible from an X-ray alone. This one experience should encourage a routine X-ray examination of all cases of suspected brain abscess.

Together with the experimental work of Dandy,[19] moreover, in the injection of air into the ventricles, it should, in the author's opinion, encourage after evacuation the injection of air in different positions of the head through the drainage tube, followed by a routine X-ray examination in different positions of the head, to ascertain whether complete evacuation of the abscess has or has not occurred. as Muck [20] has demonstrated, the size of the abscess changes with alteration in the position of the head while in the sitting position.

CASE NO. XXX.

J. B.: Roentgen Ray Diagnosis. Male, 42 years of age.

Patient admitted to hospital in stuporous condition; had had earache for several days; no history of discharging ear. When brought to hospital was suffering with pain in left ear. No temperature; slow pulse. Lumbar puncture showed almost clear fluid; globulin, ++, and high cell count—over 200 per field; many polynuclears; direct smears showed no organisms. Slight double Kernig; no marked neck rigidity. Cleared mentally after puncture, but unable to talk. When seen two days later was in deep coma; Cheyne-Stokes respiration. Temperature, 99-100; pulse, which had been 60, had risen to 160. O. E., negative. Profuse discharge from left ear.

X-ray showed shadowy area, semi-spheroidal in shape, directly above external auditory canal, the straight line of its base being nearly vertical to the external canal although slightly in front of it. The anterior portion was oval.

AUTOPSY AND REPORT BY DR. HARRISON S. MARTLAND.

On removing calvarium bone quite normal; the course of middle meningeal arteries had produced deep grooves in calvarium and there were a few small, rarefied areas in the bone. Dura normal over the top of cerebral hemispheres. On deflecting dura, which was tense, brain showed very marked picture of compression. Convolutions markedly flattened and sulci thin. The whole brain appeared dry. Small amount of subarachnoid yellowish exudate over superior surface of both cerebral hemispheres, mainly confined to regions near the longitudinal sinus and over the course of the middle cerebral arteries.

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[19] Dandy, W. E.: Localization or Elimination of Cerebral Tumors by Ventriculography; *Surgery, Gynecology and Obstetrics*, April 20, 1920.

[20] See Note 35, Chapter IV, p. 57.

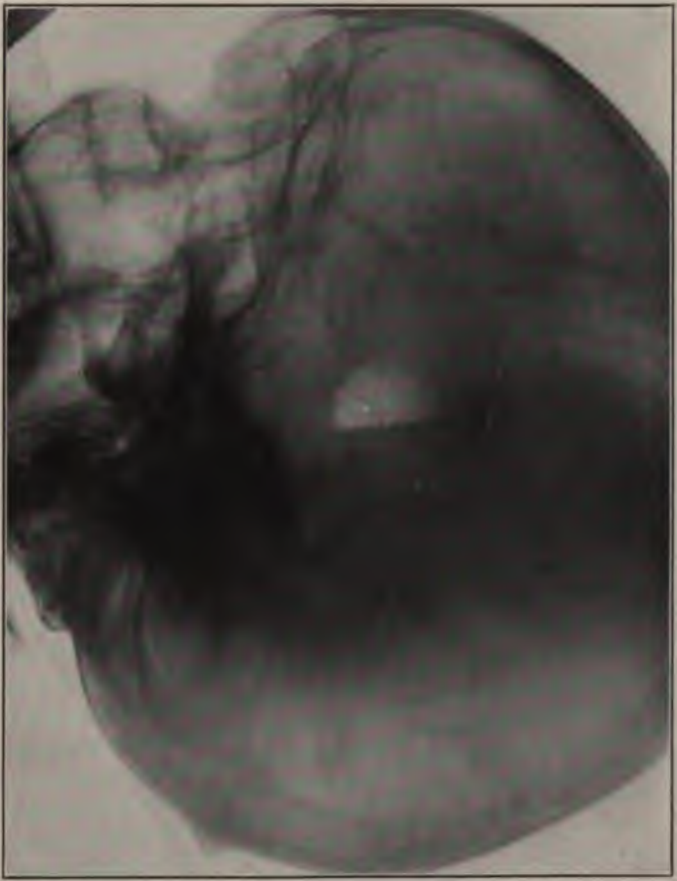

FIG. 38.—X-ray photograph showing gas of anærobic origin in cavity of temporo-sphenoidal lobe abscess. X-ray taken with patient in recumbent position. Straight line at base in "shadow" shows level of pus in abscess cavity —the gas filling the upper (anterior) portion of cavity. Note typical location of cavity of adjacent temporo-sphenoidal lobe abscess as shown by the air filling its anterior one-third.

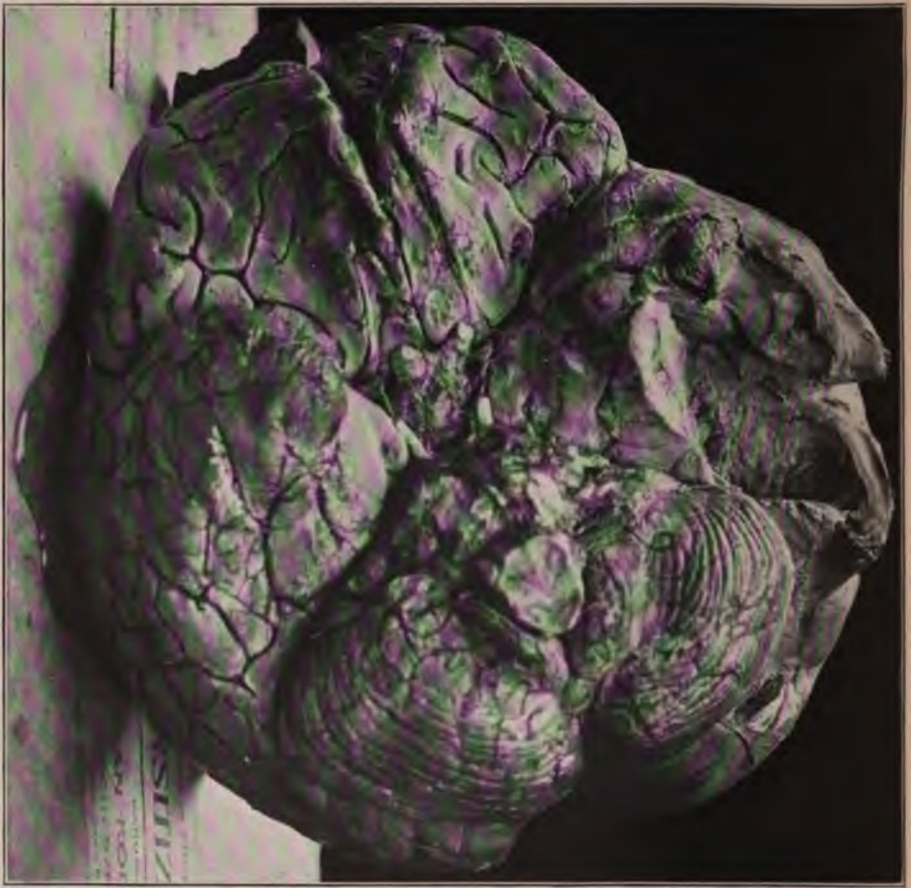

FIG. 39.—Encapsulated adjacent temporo-sphenoidal lobe abscess. Lower surface of brain removed, exposing abscess cavity, the anterior third of which was filled with gas of bacterial formation, and produced a distinct shadow in the X-ray picture. (See Fig. 38.)

On removing brain left temporo-sphenoidal lobe was tightly compressed into left middle fossa and there were present recent fibrinous adhesions between inferior surface of lobe and posterior surface of left petrous portion of temporal bone. On breaking up these adhesions in removing brain, about 40 cc. of creamy, greenish-yellow pus escaped, and several large bubbles of gas.

The large abscess cavity occupied two-thirds of left temporo-sphenoidal lobe; anterior end rounded, situated 3 cm. distant from anterior tip of lobe, opening 1 cm. in size on inferior surface of lobe. Total antero-posterior diameter of cavity 6 cm.; lateral width, 4 cm., and height, 2.5 cm. Inferior wall of abscess in thinnest place about 4 mm. thick, and in thickest areas about 1 cm., this being the distance separating the abscess cavity from the cortex of the brain. (Fig. 39.)

Abscess cavity showed wall or capsule entirely surrounding it which had internal grayish-white layer uniformly about 1 mm. in thickness; clinging to its inner surface were purulent shreds which gave it a shaggy appearance; outer part of wall reddish in color, about 2 mm. thick, making a total thickness of 3 mm. Surrounding brain substance was slightly œdematous. Dura immediately over attic showed a small perforation, circular in shape, about 0.5 cm. in size, with necrotic and blackened edges; roof of attic, necrotic; attic, middle ear, and mastoids filled with fluid pus.

Cultures from abscess showed rapid formation of gas. Type of anærobe not determined. Pus had distinctive odor, similar to that found in gangrene of the lung, which is characteristic of anærobic infection.

# CHAPTER XII.

# DIAGNOSIS OF CEREBELLAR ABSCESS.

Surgically considered, the cerebellar hemispheres are largely "silent areas," as they can be extensively destroyed without producing outspoken clinical manifestations. Cerebellar abscess, therefore, usually is much more difficult to localize than temporo-sphenoidal lobe abscess, because in the majority of cases there is an absence of the symptoms which justly may be regarded as pathognomonic, especially during the early stages. Even when the abscess has attained considerable size, if its growth is slow it frequently is not associated with any symptoms that properly may be regarded as pathognomonic. This absence of localizing manifestation is due largely to the compensation in function which is established in the cerebellum more completely and with greater rapidity than in any other portion of the central nervous system.

However, at any time during the growth of the abscess, symptoms may develop which may be sufficient to localize the suppuration in the cerebellum; of these, spontaneous vertical nystagmus, spontaneous pointing deviation, falling, commanding vertigo, lateral deviation of the eyes, cerebellar attitude, and paresis of the arm and leg of the homolateral side are the most common. Unfortunately, none of these symptoms generally are present until symptoms of compression also have appeared. Consequently, during the early stages, when surgical interference is most promising, the diagnosis in the majority of cases must rest, not on the pathognomonic symptoms, but on the ability of the surgeon to combine (*a*) the manifestations of cerebral suppuration with (*b*) the probability of the suppuration being in the cerebellum (because of the existence of a suppurative disease of a structure by way of which the cerebellum usually is invaded), with (*c*) the exclusion of other possible sites of cerebral abscess, and (*d*) *slight symptoms of impaired cerebellar function,* into enough of a localizing cerebellar picture to warrant exploration.

### LOCALIZING VALUE OF A KNOWN LABYRINTHITIS OR LATERAL SINUS THROMBOSIS.

Post-mortem examinations show that the path through which the cerebellum is invaded is by the labyrinth in 44 per cent. of the recorded cases, and by the perilabyrinth—caries of the petrous bone—in 18 per cent. more, making a total of 62 per cent. of all the recorded cases. Consequently, in the presence of a suspected localized cerebral suppuration, a non-reactable labyrinth is presumptive evidence that the abscess is situated in the cerebellum. This is a clinical fact of the highest cerebellar localizing value. In several instances the author has made a diagnosis of cerebellar abscess—the patient being in deep coma—from the presence of chronic running ear, a mild associated papillœdema and inability to induce a nystagmus by the injection of cold water into the affected ear, the absence of reaction showing a dead labyrinth.

Again, post-mortem evidence and clinical experience both demonstrate that another large proportion of all the cases of cerebellar abscess—33 per cent.—is secondary to a lateral sinus thrombosis.[1] While a lateral sinus thrombosis *may* cause an abscess in the cerebrum, the proportion of such cases is very small. Consequently, with a suspected brain abscess the presence of a known lateral sinus thrombosis is at least suggestive that the abscess is in the cerebellum.

### Symptoms More or Less Characteristic of Cerebellar Abscess.

Unlike temporo-sphenoidal lobe abscess, cerebellar abscess rarely presents in the early stages any symptoms which are absolutely pathognomonic; but careful and repeated examinations usually will elicit one or more symptoms which are of more than merely suggestive value. These may continue throughout the disease, or, what is more frequent, they may rapidly disappear. This is especially true of vertical nystagmus and lateral deviation of the eyes, both of which, when present, are definite indications of cerebellar involvement; although the former—vertical nystagmus—may occur from irritation of the cerebellum from a protective meningitis, the irritating cerebrospinal fluid in the basal cisterna originating from a distant abscess situated in the cerebrum.

*Classification:*

Cerebellar symptoms may be divided into

I—Manifestations of Impaired Cerebellar Functions;
  - (a) *asynergia, or inco-ordination of homolateral side—adiadokokinesis;*
  - (b) *homolateral "cerebellar paresis";*
  - (c) *forced cerebellar attitude;*
  - (d) *cerebellar ataxia;*
  - (e) *hypermetria.*

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[1] See Appendix II, Cerebellar Abscess.

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II—MANIFESTATIONS OF INVOLVEMENT OF CEREBELLAR PORTIONS OF THE VESTIBULAR APPARATUS;
  (a) *spontaneous* or
  (b) *induced abnormalities* of (1) *nystagmus*, (2) *vertigo*, (3) *pastpointing*, and (4) *falling*.

III—MANIFESTATIONS FROM DIRECT PRESSURE;
  (a) *cranial nerve symptoms—9th, 10th, 11th, and 12th;*
  (b) *motor pyramidal tract symptoms;*
  (c) *sensory tract symptoms;*
  (d) *sympathetic nerve symptoms—pupillary.*

IV—MANIFESTATIONS OF TRANSMITTED PRESSURE FROM INDUCED INTERNAL HYDROCEPHALUS.

## I—MANIFESTATIONS OF IMPAIRED CEREBELLAR FUNCTION EXCLUSIVE OF THE VESTIBULAR TRACT.

A better understanding of the clinical manifestations of cerebellar abscess may be obtained by recalling a few physiological facts about the cerebellum:

(A) The cerebellum is neither a motor nor a sensory, but may be regarded in a broad sense as a reflex organ, as it is by way of the cerebellum that certain sensory—peripheral—impulses influence muscular movement. The cerebellum is "related through its various lobules to the various centers in the cerebrum and the tonus centers in the medulla, in the same way that a posterior root ganglion —sensory—is related (in a motor sense) through its motor cells in the anterior horn of the cord to a certain muscle complex." [2] While it does not in any way originate motion, the cerebellum exercises a synchronizing influence on muscular movements of the same side, largely by reacting to centripetal impulses; consequently, it acts as (a) a co-ordinator, (b) a regulator, and (c) an inhibitor.

In experimental ablation of the cerebellum the primary outstanding symptoms are: (1) interference with the proper performance of certain voluntary muscular movements of the trunk or extremities, depending upon the particular part of the cerebellum attacked, this interference affecting chiefly the same side as the experimental removal; and (2) an associated alteration in the muscles in a state of rest, especially during the early stages after the ablation. Experimental ablation of the cerebellum thus causes: (a) homolateral muscular asthenia or lack of strength; (b) homolateral muscular atonia or loss of tone; (c) homolateral muscular astasia or muscular unsteadiness—this being a combination of tremor, titubation (tottering) and rhythmical oscillatory movements

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[2] Myers, I. L.: Cerebellar Localization; *Transactions of the Section on Nervous and Mental Diseases, A. M. A.*, 1916, p. 92.

—and (d) hypermetria, or lack of proper muscular inhibition. Whether the hypermetria is part of the general muscular disturbance, or exists as an independent disturbance is not at yet fully established.

All these cerebellar functions are intimately interwoven and probably all are parts of the disturbance of one function, muscular synergia—active sympathy or co-ordination.[3] Consequently, a study of the clinical manifestations of cerebellar abscess results in all the symptoms being divided into: (a) disturbance of co-ordination, viz., asynergia, or inco-ordination, of which adiodokokinesis and ataxia are the best known; (b) disturbances of muscular tone, or atonia—rhythmic oscillations, tremors, or forced positions; (c) disturbances of inhibition of voluntary muscular movements—hypermetria.

(B) In all cerebellar destructive lesions there are two stages; (1) the stage of exaltation of function, in contradistinction to the effect of invasion of the cerebrum, in which a suppurative process always is accompanied by a period of inhibition of function—such as the transient aphasia and hemianopsia of temporo-sphenoidal lobe abscess. On the other hand an invasion of any area of the cerebellum is associated with exaltation of function—such as cerebellar convulsion or a sudden lateral deviation of the eyes.[4, 5]

As the cerebellum is of low genetic origin, compensation from injury is easily established; consequently, following the stage of exaltation, there is (2) the stage of compensation by the restoration of the functions in those muscular movements and position ordinarily employed, whatever impairment of function that may remain being manifest only on attempting little-used movements—such as balancing on one foot—or on the induction of a reflex (as an induced nystagmus).

As cerebellar abscess usually develops slowly, these two stages frequently are present at the same time and it is impossible to distinguish one from another. Although at a given moment there may be outspoken symptoms of cerebellar deficiency, the establishment of compensation may cause them rapidly to disappear, with the result that at the time of examination, the composite of the two stages may give little or no clinical manifestation of impaired cerebellar function. It is the early establishment of compensation which makes the diagnosis of cerebellar abscess so difficult and at the same time makes an *altered or transient impairment of cerebellar function* of diagnostic importance.

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[3] Mills, Charles K., and Weisenburg, Theodore H.: Cerebellar symptoms and Cerebellar Localization; *Transactions of the Section on Nervous and Mental Diseases, American Medical Association*, Atlantic City, June 23-26, 1914, p. 107.

[4] Case XXXI, W. J., Chapter Twelve, p. 203.

[5] Meyers, I. L.: *Loc. cit.* ("The phenomena of cerebellar deficiency are accordingly to be interpreted as phenomena of hyperfunctional and not hypofunctional activity.")

### PARTIAL OR COMPLETE OBLITERATION OF A SYMPTOM THE RESULT OF CEREBELLAR COMPENSATION.[*]

While it may be impossible to distinguish any one symptom as the direct result of compensation, the obliteration of the symptom by compensation should be regarded as the important thing in an attempt to interpret a vague cerebellar manifestation in the case of a suspected cerebellar abscess.

**Cerebellar Paralysis.**—The cerebellum undoubtedly has control of the co-ordination of the muscular movements of the body as well as of the tonicity of the individual muscular group. It thus acts as a "reinforcing" station. For this reason the presence of a lesion within the cerebellum may produce an apparent muscular paralysis. Unless the pyramidal tract is invaded the paralysis is never complete and affects only the side of the body on which the lesion is located. Homolateral, incomplete paralysis, therefore, is an aid to the localization of an abscess. When a true motor paralysis occurs in a cerebellar lesion, it is due to the effect of transmitted pressure in the pyramidal tracts and not to involvement of the cerebellum itself.

**Cerebellar Ataxia.**—Cerebellar ataxia is made up of three parts: muscular asthenia—loss of strength, or debility; muscular atonia—a lack of tone; and muscular astasia, the latter being a combination of titubation, or tremor, with rhythmical oscillatory movements, and hypermetria, a lack of inhibition.

Ataxia is but infrequently seen in cerebellar abscess because long before it has become of sufficient severity to be of diagnostic assistance, marked symptoms of compression have developed. The author is of the opinion, however, that a careful study of the "vertigo" so frequently classed as labyrinthine, chiefly because of the presence of a non-reactable labyrinth, would disclose evidences of cerebellar ataxia enough to warrant at least a suspicion of cerebellar involvement.

**Hypermetria—"Failure of Inhibition."**—One of the functions of the cerebellum is inhibition of muscular movement. Whether this is intimately related to, or really is a part of the other functions of this part of the brain—synergia, tonia, co-ordination, and so on—has not been definitely established.

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[*] Luciani, Luigi: *Human Physiology*, 1915, Vol. III, Muscular and Nervous Systems, p. 481. ("Functional compensation is the purposive and voluntary acts by which the effects of deficient or lost cerebellar innervation is abrogated.")

Rossi, Gilberto: *Archivio di fisiologia*, 1912, Vol. X, p. 251. (The cerebrum also assists in the establishment of compensation, as "The immediate effect of hemi-extirpation of a dog's cerebellum is diminution of excitability in the motor cortical area of the opposite hemisphere.") Quoted by Luciani in *Human Physiology*, 1915, Vol. III, p. 483.

In one of the author's cases a test of the past-pointing of the homolateral side revealed the inability of the patient to control the backward movements of his arm. He invariably brought the arm so far back that it struck the bar at the top of the bed with sufficient force to cause pain—an evidence of hypermetria. There was also a lack of co-ordination laterally, yet he could touch the fingers together fairly well. On operation it was found that the abscess was 3 cm. inward and upward, probably involving the arm center; therefore, the asynergia, or inability to synchronize the movements of the joints and muscles, was in the arm or shoulder (probably in the latter).

**Cerebellar Convulsions.**—Experimentally, on the removal of certain parts of the cerebellum there follow oscillatory or contractile movements of the homolateral side, resulting in a to and fro movement of the head or a forced position of the trunk. On superficial cauterization of the same cerebellar region, however, a similar condition occurs on the *opposite* side of the animal.[7] Whether this phenomenon is due to irritation or to the removal of inhibitions is still in dispute.

While convulsions are of but infrequent occurrence in tumors of the posterior fossa,[8] they are frequently associated with cerebellar abscess.

**Forced Cerebellar Attitude.**—Lying on one side in a semi-flexed attitude is frequent in cerebellar abscess during the stage of compression. Its presence prior to this stage is of the first diagnostic importance even though it be imperfectly developed. Likewise, a lateral deviation of the eyes occurring prior to compression should be regarded as a forced cerebellar position.

The lateral deviation of the eyes observed in Case No. XXXI, W. J., Chapter Twelve, p. 203, following a convulsion was a manifestation of the "sustained tonicity" which is characteristic of cerebellar convulsive movements. In several other of the author's cases a forced position on one side—has been the guiding diagnostic manifestation.

**Speech Defects.**—A dysarthria—a scanning or ataxic speech—is a frequent manifestation of cerebellar abscess. It is one of the many manifestations of muscular inco-ordination.

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[7] Ferrier: See Luciani, *Human Physiology*, Vol. III, Muscular and Nervous Systems, p. 434.

[8] McRobert, R. G., and Feinier, Laurent: Cerebellar Fits; *Archives of Neurology and Psychiatry*, March, 1921, Vol. V, p. 304.

### II—VESTIBULAR MANIFESTATIONS.

Unfortunately, the same vestibular syndrome—spontaneous nystagmus, past-pointing, and falling—which always accompanies acute labyrinthitis, is also produced by an acute, destructive lesion of the cerebellum, and as cerebellar abscess in a large proportion of the cases is a complication of a suppurative labyrinthitis, differentiation is thereby rendered difficult. To be fully successful the attempt to differentiate must be made immediately upon the appearance of any one of the symptoms common to both, for while theoretically there are many points of difference between the vestibular manifestations of a suppurative labyrinthitis and an intracranial involvement[9] (the falling, for instance, in a labyrinthine involvement being influenced by a change of position of the head while in a cerebellar lesion it is not), in actual practice such is the case only when the cerebellar lesion is *acute, limited* and *destructive.* In cerebellar abscess, however, the slow growth may allow ample time for the reestablishment of compensation, with the result that at the time of examination either no vestibular symptoms may be present at all or they will be so modified as to have lost their distinctive cerebellar characteristics.

If assistance in the early diagnosis of cerebellar abscess is to be obtained from the vestibular manifestations it can be accomplished only by repeated observations, and recording *at the time of observation*, all data as well as all vestibular manifestations normal and abnormal, for in this way alone a changing character will be appreciated. If to-day no nystagmus is present in the recumbent or in the erect position, and to-morrow there is a slight nystagmus to the right in the same position, and on the following day it is again absent or has changed to a slight nystagmus to the left, the record of the three days may make an early diagnosis possible, while failure to record one or the other manifestation at the moment of observation may result in doubt.

#### DIFFERENTIAL VALUE OF MODE OF ONSET.

In acute labyrinthitis the onset of the suppuration is sudden and complete, the limited area of the bony capsule causing a complete inhibition of function. The spontaneous nystagmus, past-pointing, and falling, all appear at the same time; and as compensation is established the symptoms individually and gradually disappear, first the nystagmus, then the falling, and last of all the past-pointing. In cerebellar abscess, on the other hand, there is a disproportion in the complete onset and the progressive disappearance of the symptoms. Consequently, an incomplete onset is of especial importance in diagnosing a cerebellar lesion.

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[9] Moulouquet, A.: Cerebellar Abscess of Aural Origin; *The Lancet*, 1921, I, p. 283. (States "in cerebellar abscess the disturbances are more equilibrating than vertiginous, while in labyrinth disease it is the vertiginous character which predominates.")

### SPONTANEOUS NYSTAGMUS.

The spontaneous nystagmus of cerebellar abscess is apt to be transient and of changing character during the early stages. It is also of a somewhat different type from that of involvement of the labyrinth. The nystagmus of cerebellar abscess, being due to asynergia of the ocular muscles, is in consequence more or less oscillatory in nature, while the nystagmus of vestibular origin is rhythmic.[10] Cerebellar nystagmus usually is toward the side of the lesion, with a vertical tendency. Too much attention should not be paid to this, however, as the ease with which the cerebellum establishes compensation not only may alter the character of the nystagmus but may eliminate it altogether.

### LATERAL DEVIATION OF THE EYES.

Lateral deviation—the cerebellar component of a nystagmus—generally occurs when compression or destruction is sufficient to cause an abolition of the cerebral control. The author is of the opinion that compression sufficient to cause coma and lateral deviation is much more easily induced in cerebellar than in cerebral abscess. When an otitis media is the primary focus of a brain abscess the appearance of a lateral deviation of the eyes should be regarded as evidence that the posterior fossa is the site of the infection since an adjacent temporo-sphenoidal lobe abscess can not cause a spontaneous lateral deviation of the eyes.

### VERTIGO, SPONTANEOUS FALLING AND SPONTANEOUS PAST-POINTING.

The combination of vertigo and falling with marked nystagmus so frequently seen in cerebellar pontine angle tumors is rarely present in the early stages of cerebellar abscess, or if present is so complicated by disease of the labyrinth as to make interpretation difficult.

(a) *Vertigo:* While a symptom of disease of the labyrinth without intracranial involvement, vertigo frequently is the only outstanding indication of cerebellar abscess as it occurs as a marked symptom in about one-third of the cases.[11] Contrary to the generally accepted view vertigo may be entirely absent in the early stages of the disease.[12] Spontaneous vertigo may be of localizing value, as inability to alter the vertigo by the application of caloric and rotating tests is presumptive evidence that the lesion is in the cerebellum.

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[10] Wilson, J. Gordon, and Pike, F. H.: Mechanism of Labyrinthine Nystagmus and its Modification by Lesions in the Cerebellum and Cerebrum. An Experimental Investigation; *Oxford University Press*, Henry Frowde, 1914. Also *Journal American Medical Association*, December 18, 1915, Vol. 65, p. 2156.

[11] Dench, Edward Bradford: Otitic Brain Abscess; *Reprint from American Journal of Medical Sciences*, November, 1907, p. 3.

(b) *Falling:* Physiologically, it would be expected that in abscess of a cerebellar hemisphere falling when present would be toward the side of the lesion and would not be influenced by changing the position of the head. Due to the establishment of compensation by the cerebellum, however, the direction and the type, and even the presence of falling may promptly be altered or abolished. In one of the author's cases of adjacent cerebellar abscess the falling was backward and away from the lesion; but so many cases are recorded in which the falling is similarly paradoxical and even influenced by a change in the position of the head,[13] that, like nystagmus, it must lose its clinical localizing significance. When the vermis primarily is involved—a rare situation in cerebellar abscess except in metastatic cases—the falling probably would be backward.

(c) *Spontaneous Past-Pointing Deviations:* Spontaneous pointing deviations occur in the early stages of brain abscess, but abnormalities are so frequent that they cannot be considered as symptoms of great localizing value.

## INDUCED ABNORMAL VESTIBULAR REACTIONS.

Too much reliance has been placed upon the value of one complete examination of induced vestibular reactions in locating the presence of, or eliminating the cerebellum as the site of an abscess, as clinical experience has demonstrated that only by repeated tests can the functioning of the cerebellum be shown to be in perfect order.[14]

The tests must be made before the patient becomes dulled. The author's experience has been that all patients—even small children—can be subjected to the tests provided the examiner will devote sufficient time and patience to making them.

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[12] Case XII, W. K., Chapter Four, p. 60.

[13] Rohardt, W.: Ueber Zeige und Fallreaktionen bei Kleinhirnkranken; *Zeitschrift für die ges. Neurologie und Psychiatrie,* 1919, Vol. 49-50, p. 167.

[14] *Reported Cases of Cerebellar Abscess Giving Spontaneous and Induced Vestibular Reaction and Demonstrating their Changing and Bizarre Character.*

Hohngren, G.: Opererad och läkt lillhjärnabscess med skada a ett ave Barany's centra; *Svenska Läkaresällskapets Handlingas,* 1916, Vol. XLII, p. 516.

Jones, I.: Equilibrium and Vertigo; 1918, p. 369 (Case by Fisher).

Hill, Frederick Thayer: Report of a Case of Cerebellar Abscess Following Shell Wound of Skull and Showing No Symptoms for Period of Five Months; *Military Surgeon,* September, 1919.

Rohardt, W.: Ueber Zeige and Fallreaktionen bei Kleinhirnkranken; *Zeitschrift für die ges. Neurologie und Psychiatrie,* 1919, Vol. 49-50, p. 167.

Jessaman, L. W.: Brain Abscess as a Complication of Acute Infection of Nasal Accessory Sinus; *Laryngoscope,* March, 1920, Vol. XXX, No. 3, pp. 147-149.

Martin, H. H., and Crowe, S. J.: Lateral Sinus Disease—Case Report; *Laryngoscope,* December, 1920.

### CHANGING CHARACTER OF ABNORMAL SPONTANEOUS OR INDUCED VESTIBULAR MANIFESTATIONS.

The author's experience leads him to believe that there is no set formula for the diagnosis of cerebellar abscess by abnormalities of the vestibular reactions alone, but that the induced reactions, like the spontaneous manifestations, are apt to be of a very changing nature, especially during the early stages of the disease. This changing character he has come to regard as highly suggestive of cerebellar involvement.

In one of his cases[15] the only symptom present at the time of examination was an abnormal falling; there was no spontaneous nystagmus, and aside from the falling, the vestibular reactions apparently were normal; but suddenly the patient had a convulsion, followed by a lateral deviation of the eyes.

Small, acute, and destructive lesions, such as hemorrhages involving the so-called "vestibular pathways," will produce symptoms or give localizing manifestations; but cerebellar abscess is a gross lesion which at the time of examination generally has progressed slowly to the point of giving rise to an increased intracranial pressure, not only of the posterior fossa but also of the whole intracranial contents. During the slow, progressive growth of a cerebellar abscess the cranial contents have had ample time for readjustment—the establishment of compensation. This compensation the vestibular apparatus—one of the most primitive of the special senses—is especially able readily and easily to accomplish; with the result that at the time of examination no diagnostic abnormalities of induced vestibular reactions may be present, while the *vestibular manifestations of the increased intracranial pressure overshadow and obscure all others.* For several reasons not yet fully explained an increase of intracranial pressure interferes with the reactability of the intracranial portion of the fibres of the vertical canals of both ears long before the fibers to the horizontal canals are affected. Whether this affection occurs on the auditory canal itself, or in the intracranial portion, is not definitely established, but it is probably in the latter.

### DIAGNOSIS OF INCREASED INTRACRANIAL PRESSURE, ESPECIALLY OF THE POSTERIOR FOSSA, BY INDUCED VESTIBULAR REACTIONS.

The outspoken vestibular manifestation of increased intracranial pressure—absence of reactability of the vertical canals of both ears to the cold caloric—first described and properly interpreted by the author,[16] while an early manifestation of increased intracranial pressure prevents an accurate localization by the vestibular reactions alone. It is of the greatest value, however, in the early diagnosis of increased intracranial pressure as it appears long before and independently of papillœdema.

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[15] Case XXXI, W. J., Chapter Twelve, p. 203.

Its assistance in the diagnosis of cerebellar abscess by demonstrating an increased intracranial pressure cannot be over-emphasized. As the result of many vestibular examinations and subsequent lumbar punctures for diagnosing obscure nervous diseases, the author testifies that the vestibular manifestations of increased intracranial pressure are uniformly the earliest symptom present and consequently the most valuable as yet known.

### Cerebellar Cortical Localization in Relation to Vestibular Reaction.

Horsley and Clark [17] demonstrated that the cerebellar cortex contains no areas which are irritable by the faradic current, such as exist in the motor areas of the cerebrum. When muscular contraction occurs from such irritation it is due to involvement of the deep ganglia. There is, however, considerable experimental and clinical evidence to substantiate Barany's contention [18] that the cortex of the cerebellum contains areas the destruction of which—or pressure upon them—causes alteration in the ability to execute certain voluntary movements properly.

### Induced Vestibular Manifestation from Pressure upon the Cerebellar Cortex.

The author has observed that pressure from a protective exudate over the anterior surface of the cerebellum opposite the internal auditory meatus uniformly results in (a) an immediate reduction of the duration of the obtainable nystagmus from the horizontal canals by turning, and (b) a great reduction, or complete absence, of irritability of the superior vertical canals of both sides on the application of the cold caloric,[19] the latter from increase of the intracranial pressure. If this interpretation of the clinical observation is correct, the vestibular symptom complex—viz., reduction by about 50 per cent. of the normally obtainable nystagmus from pressure on the cerebellar cortex opposite the internal auditory—should render possible an early diagnosis of intrapiarachnoid abscess of the anterior surface of the cerebellum (the most frequent site of intrapiarachnoid abscess from labyrinth suppuration).

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[16] Eagleton, W. P.: Decompression for Relief of Disturbances of the Auditory Apparatus of Intracranial Origin—Report of Three Cases with a Previously Undescribed Aural Condition; *Transactions American Otological Society*, June 10, 1912, Part III, p. 564. Also *Laryngoscope*, 1913, Vol. XXII, p. 592.

[17] Horsley and Clark: On the Intrinsic Fibers of the Cerebellum, its Nuclei and its Different Tracts; *Brain*, 1908, Vol. XXXI, p. 138. (Quoted by Luciani, in Human Physiology, 1915, Vol. III, Muscular and Nervous Systems, p. 484.)

[18] Barany, R.: The Vestibular Apparatus and the Central Nervous System; *Laryngoscope*, 1912, Vol. XXII, p. 81, demonstrated that the cooling of "small areas of the cerebellar cortex situated directly behind the attachment of the external ear," results in a spontaneous past-pointing of the arm *outward*, from, he believes, a paralysis of the inward pointing centre, while cooling an adjacent area causes a spontaneous past-pointing *inward*.

### III—LOCAL SYMPTOMS FROM DIRECT PRESSURE PRESUMPTIVE OF CEREBELLAR ORIGIN.

(a) *Hyperesthesia of the face and diminution of the corneal reflex on homolateral side—fifth nerve involvement;*

(b) *Deviation of tongue—twelfth nerve involvement.*

Deviation of the tongue may be due to direct pressure or it may be due to involvement of the nucleus of the twelfth in the medulla or of the supra-nuclear pathway. In the former case the tongue will deviate away from the side of the lesion and in the latter toward it.

Absence of corneal reflex probably is due to direct pressure as it occurs on the side of the lesion. It probably is caused by pressure of the upper fibres of the sensory root against the firm dural opening where the root passes out of the posterior fossa into the Meckel's space.

On the involvement of the ninth—the glosso-pharyngeal—the author's only experience was in one case, in which the patient complained of difficulty in swallowing.

The respiratory and circulatory disturbances of the tenth hardly can be expected to be elicited in cerebellar abscess.

### PUPILLARY DISTURBANCES.

The dilatation of the pupil so frequently seen in cerebellar abscess is due more to action of the toxemia upon the sympathetic centers than to any direct effect of the abscess.

### GENERAL SYMPTOMS SUGGESTIVE OF CEREBELLAR INVOLVEMENT.

(a) *Suboccipital tenderness and rigidity of neck;*

(b) *Yawning;*

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[19] (a) Eagleton, W. P.: Aural Manifestations in Surgical Intracranial Lesions; *Address before Clinical Congress of Surgeons,* Philadelphia, Oct. 25, 1921. (b) Eagleton, W. P.: Value of Vestibular Manifestations in Surgical Intracranial Conditions—with Report of Four Cases; *Address Annual American Laryngological, Rhinological and Otological Society,* Atlantic City, June, 1921.

(c) *Rapid loss of flesh;*
(d) *Alteration in knee jerks;*
(e) *Projectile vomiting;*
(f) *Psychic disturbances and cerebellar fits.*

**Suboccipital Tenderness.**—Cushing and Grey [20] have called attention to the greater frequency of suboccipital tenderness in cerebellar than in cerebral tumors. The author's experience would lead him to believe that this is also the case in cerebellar abscess.

**Yawning.**—Although yawning frequently is present in suppurative disease of any part of the brain, in the author's experience it is more marked in cerebellar abscess than in abscess in other regions.

**Rapid Loss of Flesh.**—Emaciation in cerebellar abscess is much more rapid than in suppuration in other parts of the brain.[21] It is partially due to the effect of the cerebral suppuration in disturbing the general metabolism. In cerebellar cases, however, there is added more or less of a specific action from the disturbance of the trophic reactions presided over or reinforced by the cerebellum, which results in a rapid emaciation. When the abscess becomes completely encapsulated and quiescent, the emaciation stops. In the presence of a probable cerebral suppuration the author has come to regard rapid emaciation as suggestive of cerebellar abscess. Experimentally, injury to the cerebellum results in "retarded growth of the cutaneous elements, particularly of the skin, and the lowering of resistance to the injurious action of external agents." [22]

**Knee Jerks.**—Alteration in the patella reflexes depends upon the involvement of the pyramidal tracts; it generally occurs on the homolateral side, but it may be bilateral.

**Vomiting.**—Vomiting is much more sudden and severe in cerebellar abscess than in abscess in other locations. In the author's experience in the presence of a suspected localized cerebral suppuration, continuous projectile vomiting is suggestive of cerebellar abscess.

**Psychic Disturbances.**—In the presence of compression there always occurs a cloudiness of the sensorium—poor perception and attention—and possibly disorientation for time and place.[23] In intracerebellar abscess the author has found a certain degree of optimism in the patient which is not warranted by his physical condition. In

[20] Grey, Ernest G.: Studies on the Localization of Cerebellar Tumors; *Reprinted from Annals of Surgery*, February, 1916.

[21] Luciani, Luigi: *Human Physiology*, 1915, Vol. III, Muscular and Nervous Systems, p. 472.

[22] Luciani, Luigi: *Loc. cit.*

[23] Mingazzirn, G.: Abscess e Tumori del Encefalo; Riv. di Patol., Nov., 1919, Part 3-4 and 5-8. Abstracted in *Archives of Neurology and Psychiatry*, 1919, Vol. 2.

one of his cases the patient, throughout his illness and even when dying, continued to insist that he felt fairly well.

### IV—SYMPTOMS DUE TO AN ACCOMPANYING INTERNAL HYDROCEPHALUS.

On account of its position in the posterior fossa, cerebellar abscess is much more frequently associated with an internal hydrocephalus from obstruction by pressure and displacement of the iter, than is an abscess in any other portion of the brain. Stupor, alterations in the reflexes, epileptiform seizures, intense and paroxysmal headaches, sub-occipital tenderness, opisthotonos, sugar in the urine, vomiting—all are frequent and all, partially at least, originate from the internal hydrocephalus; while the marked papillœdema of cerebellar abscess is, in the author's opinion, entirely of this origin, the distance of the site of the suppuration away from the optic nerve being best explainable by the induced internal hydrocephalus.

To this group belong also the sixth nerve paralysis, from pressure, as the nerve passes over the apex of the petrous base in its separate dural compartment. Third nerve paralysis probably is from pressure exerted between the internal carotid and the distended floor of the third ventricle and the intra-chiasmal cisterna.

The slight exophthalmos very rarely seen in cerebellar abscess probably originates from the pressure of the internal hydrocephalus, and the hyperesthesia of the fifth probably has a similar origin.

CASE NO. XXXI.

W. J.: Case of cerebellar abscess, illustrating vagueness of early symptoms, followed by remission of all symptoms; then sixth nerve paralysis. No localizing symptoms whatsoever until nineteenth day, when suffered from dizziness for first time. Tempero-sphenoidal lobe exploration from erroneous diagnosis. Sudden onset of marked cerebellar manifestations; several incomplete cerebellar evacuations. Changing character of vestibular manifestations. Extension of abscess.

**Autopsy.**—Multilocular abscesses of cerebellum.

**History.**—

Boy.

*March 1:* Cold, accompanied by pain in right ear, followed by discharge.

*March 12:* Mastoid operation.

*March 23:* Severe headache through forehead and back of head, thought to be due from chilling from sudden change in weather. Invasion of cerebellum undoubtedly occurred at this time.

*March 24-29:* Perfectly well.

*March 30:* General malaise and "some fever."

*March 31-April 3:* Again perfectly well.

*April 4:* Nauseated; from this date was never entirely well.

*April 5:* Sudden double vision; which caused slight feeling of unsteadiness which disappeared upon closing one eye. Nosebleed.

*April 6:* Vomited; nose bleed; felt well enough to go to physician's office for dressing.

*April 8:* Vomited; tongue coated; severe parieto-occipital headache; confined to bed. For first time looks ill.

*April 11:* Lumbar puncture; fluid clear, not under pressure; Fehling's reduced; low cell count, 5; vomited after lumbar puncture.

*April 12:* Admitted to hospital, complaining of headache, vomiting, double vision. To-day has had first attack of real dizziness (nineteen days after cerebellar involvement). "When he turns in bed feels sick at stomach." Slight aching in right shoulder and right hip. Patient thinks there is a slight weakness in right leg, "especially since having needle inserted into him"—the lumbar puncture.

**Examination.**—Memory good; has rather "dopey" manner; answers questions intelligently but in a low voice. Brightens up on being questioned; speech rather thick as though tongue were thick; patient says he has noticed change himself—that he cannot pronounce words as rapidly as he should. Thinks it is due to the thick coating on his tongue.

*April 13:* Pulse 84-90; complained of severe pain in head. Examination to-day shows slight nystagmus on looking toward extreme right (this is the first suspicion of any nystagmus since the beginning of his illness). Hears with his right ear.

*April 14:* Lumbar puncture, fluid clear. Fehling's reduced normally, cell count 57 per cm.; no organisms. Diagnosis, probable abscess of the temporo-sphenoidal lobe with capsule, because vestibular reactions apparently demonstrated increased intracranial pressure with an intact cerebellum.

*Résumé:* An absence of vestibular reaction of his vertical canal and a normal reaction to the horizontal.

**Operation.**—Temporo-sphenoidal lobe exploration; no pus but accidental tapping of the lateral ventricle. On account of the puncture going into the ventricle at a very short distance, decided that patient either had a serous meningitis, or that he had a cerebellar abscess.

Condition at the completion of the operation very bad; pulse very fast; decided not to explore the cerebellum until he had time to recover.

*April 16:* Pulse 80; has vomited several times.

*April 17:* Restless; complains of severe pains in temples. Lumbar puncture 15 cc. fluid clear, cell count 118; Fehling's reduced normally.

*April 18:* Had a convulsion. Became unconscious, following which had lateral deviation of eyes to left (away from the site of the lesion). Left eye now looks down and out. Co-ordination, right hand not so good as left, tongue apparently protrudes to the right. A Romberg.

**Operation. Cerebellar Exploration.**—*April 18:* Ligation of lateral sinus. Area of caries in the posterior portion of the petrous pyramid exposed, from which infection had entered the cerebellum. Encapsulated abscess, located at distance of 3 cm. from cortex. Slow evacuation through small exploring canula. Smear from abscess showed streptococcus and Gram positive, bacillus and sarcinæ. Operative comments: "The abscess is probably much larger than evacuated; a larger exploring canula should have been used. The abscess was apparently 'lost' before complete evacuation. Patient stood the ether very well, which is remarkable, as at the previous operation he nearly died on the table, and to-day he is in very much worse physical condition. This may be explained by the accidental tapping of the ventricle and the relief of pressure at the previous operation."

*April 21:* Evacuation; marked nystagmus on looking toward the right—toward the lesion, while previous to the evacuation he had a lateral deviation of both eyes to the left. Patient now in excellent mental condition.

*April 23:* Abscess apparently not evacuated completely. Patient in very good condition but sweats too much. Still has a marked spontaneous nystagmus, which has a tendency to be vertical in character.

*April 26:* Patient has become more and more drowsy; has nystagmus; cannot tell whether it is to the right or to the left, but it certainly has a vertical tendency up and down. No choked disc, undoubtedly because he has such a large decompression. Marked hypermetria of the right arm.

**Operation.**—Evacuation of abscess well up toward the tentorium and in the anterior portion of the cerebellum.

*May 1:* Patient vomited for first time since April 24th; recurrence of headache. No rise in temperature; pulse 82; culture from abscess streptococcus proteus.

*May 2:* Mild papillœdema of right eye; left negative. Head back, slight nystagmus to left in outer corner. Spontaneous past pointing both hands elevated slightly to left spontaneously. Head erect, no perceptible spontaneous nystagmus; spontaneous past pointing; tendency of right hand to deviate to right; left deviates to left.

*May 6:* Still indistinctness of upper margin of right disc, but much less than three days ago. Still incoördination of right arm, fingers to nose test. Rotates right hand much slower. *Spontaneous nystagmus becomes marked on changing position.*

During the whole of this time patient remained in a rather apathetic condition, saying he felt pretty well, but having a tendency to sleep, the sleep not being of a natural type.

*May 13:* Erect position; spontaneous nystagmus on looking up, but not so marked on looking to right—side of lesion, but marked on looking to left—away from lesion. Legs: touches hand with left toe, but right leg shows incoördination. Astereognosis. O. E.: right eye, indistinctness toward nasal side. Left eye may be slightly hazy above. *Pupils large in proportion to amount of light in room, but react promptly.* No Babinski, no Oppenheim. Speech slow and rather cut off—talking somewhat like a Chinaman, without proper modulations, in a monotonous tone. Repeats and fumbles over parts of words—then repeats the word and goes on again. Tongue protrudes slightly to the right side—side of lesion; when sits up sways (trunk muscles involved); slight dizziness. All signs of extension of cerebellar suppuration.

*May 14 to July 3:* Abscess evacuated on several occasions. Increasing stupor. Death.

**Post-Mortem Notes.**—Autopsy and report by Dr. F. A. Sutton.

Dura over right cerebellar area adherent. Considerable pus. The right cerebellar lobe little more than a shell. The abscess cavity extending over beyond the median line into the left lobe and almost through the cortex on the superior surface. On dissecting cerebellum there were several cavities containing from one-half to a dram and one-half of pus. The abscesses resulted in almost complete destruction of the right cerebellar lobe and practically one-half left lobe of cerebellum. The drainage was not complete because the abscess was multilocular.

Several small harder areas near the surface of the cerebellum, consisted of normal brain tissue surrounded by blood vessels. Lateral ventricles free. The third ventricle slightly distended with fluid.

## CHAPTER XIII.

## DIAGNOSIS OF FRONTAL LOBE ABSCESS.

Frontal lobe abscess is the most difficult of all types of abscess to diagnose, (1) because the symptoms usually present in acute and chronic frontal sinus or ethmoid disease closely simulate those of frontal lobe abscess; (2) because the region of the frontal lobes involved in adjacent abscess is a "silent area" and consequently there are but seldom any definite manifestations of cerebral involvement; (3) because of the small space occupied by the frontal lobe, the abscess does not attain to any considerable size before it ruptures into the ventricles or the subarachnoid space.

An examination of the recorded cases of frontal lobe abscess (see Appendix III, Frontal Lobe), reveals that a large proportion were not diagnosed during life, a sudden and fatal termination having occurred as the result of rupture of the abscess. Therefore, if the diagnosis is to be surgically useful, it must be made before the signs of compression have appeared, since a frontal lobe abscess usually does not give signs of compression, at least not until immediately before the fatal termination.

An early diagnosis of frontal lobe abscess is made possible only by a systematic method of reasoning, together with repeated and thorough examinations, with minute attention to certain small details. This is especially true if the abscess is latent and of small size. The diagnosis must depend largely upon: (1) the presence of a known point of infection which is apt to cause an abscess of the frontal lobe—in the frontal sinus, the ethmoids or the orbit; (2) the history of an acute invasion or an acute exacerbation of a chronic suppuration—tenderness, redness of the external frontal wall, orbital phlegmon, or trauma, accidental or operative—since such irregularities are more or less constantly present; and (3) the general symptoms of cerebral suppuration.

*Orbital Abscess, Osteomyelitis and Extradural Abscess—Disproportion; value of.*—The presence of any one of these conditions presupposes a possibility of a frontal lobe abscess. When associated with symptoms disproportionate to the known lesion, or with symptoms of cerebral suppuration, they call for exploration within the dura. The scar of a traumatic wound of the frontal sinus, followed by headache, should presuppose frontal lobe suppuration. Many cases of frontal lobe abscess subsequent to an injury, are on record, in which careful examinations have failed to reveal any mental disturbance or psychic effect, the only symptoms present being headache, insomnia and anorexia—general symptoms of cerebral suppuration.

*Symptoms of Cerebral Suppuration.*—There is a certain look about the patient which, to the trained eye, is very significant; the tongue is coated, the skin dry, there is loss of flesh, and the leucocyte count is high. Headache is uniformly present and in many cases is the only symptom. It frequently dates from an acute exacerbation of a chronic frontal sinusitis, and if so should be always regarded with suspicion. The headache may be continuous or intermittent, but it is of a more severe type than that which occurs with frontal sinus suppuration alone. If, to the above, is added vomiting, and if the cerebro-spinal fluid from a lumbar puncture gives a high cell count, exploration of the frontal lobe is indicated. The cerebral exploration should precede, not follow, an operation upon the accessory sinuses.

On the other hand if an extradural abscess is accidentally located at the time of a sinus operation, the existence of an associated intradural abscess would be suggested.

*Convulsions.*—In the author's opinion convulsions are positive evidence of intracranial involvement. While it is possible that a convulsion may occur from a protective piarachnoid accumulation, when the sinus has been opened or free nasal drainage has been established, the occurrence of convulsions should be regarded as distinct evidence that the disease has extended beyond the accessory sinuses themselves.

The cases of cerebral symptoms which have been cleared up by operations on the frontal sinus are due to "serous meningitis" of toxic origin.

*Diabetes Insipidus.*—Foerster [1] calls attention to the appearance and development of diabetes insipidus in serous meningitis. As it cannot occur from an acute sinus disease alone, it should be classified among the symptoms of cerebral origin.

### SYMPTOMS OF CEREBRAL COMPRESSION.

*Coma.*—The coma of frontal lobe abscess is somewhat different from the coma seen in general cerebral compression. It simulates normal sleep more than the coma of suppuration in other parts of the brain.[2]

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[1] Foerster: Munich. med. Wochenschrift, 1918.

In one of the author's cases this so-called "frontal-lobe coma" was present for several weeks. At the autopsy one of the ventricles was found to be distended with pus.

*Papillœdema.*—If papillœdema were caused by direct pressure it would be of frequent occurrence in frontal lobe abscess. Such is not the case, however. In frontal lobe abscess papillœdema is rarely present.[3, 4, 5]

An examination of the records of over one hundred cases of frontal lobe abscess shows only three to have exhibited definite nerve head changes, all associated with symptoms of compression. The almost uniform absence of papillœdema in frontal lobe abscess is due to the fact papillœdema is largely dependent upon an obstruction of the central cerebro-spinal fluid circulatory system.

The situation of a frontal lobe abscess causes no such obstruction. It is not unusual, however, to have papillœdema appear immediately upon the evacuation of the abscess, which demonstrates that the trauma of the operation furnishes a factor necessary to the production of papillœdema, although the increased intracranial pressure had been relieved.

### POST-NEURITIC RETROBULBAR NEURITIS.

The author has seen one case of the retrobulbar neuritis described by Kennedy[6] in a frontal lobe tumor; it is not recorded in any case of abscess of the frontal lobe.

### LOCALIZING SYMPTOMS.

(a) *Loss of Smell.*—Loss of the sense of smell on the affected side probably occurs very frequently. If present it is of distinct diagnostic value.

(b) *Aphasia.*—In frontal lobe abscess aphasia occurs only when the abscess has attained considerable size. At first it is apt to be transient, due probably to an extension of œdema to the Island of Reil. In many of the recorded cases of frontal lobe abscess of the left side, associated with paresis or paralysis of the arm of the opposite side, motor aphasia has been the first symptom.

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[2] Millan, G.: Le Coma Frontal; *Paris Medical*, 1920, Vol. X, No. 40, page 256.

[3] Denker, A.: Rhinogener Frontallappenabszess: *Archiv für Laryngol. und Rhinol.*, 1900, X, S. 411. Blurring of the left disc; pulse 55-60.

[4] Faunz, M.: Rhinogener Hirnabszess; *Archiv für Laryngol. und Rhinol.*, 1902-03, XIII, S. 427. (Venous hyperæmia of the outer fundus; had compression, pulse being 60-66.)

[5] Jessamon, L. W.: Brain Abscess as a Complication of Acute Infection of Nasal Accessory Sinuses; *Laryngoscope*, 1920, Vol. 30, p. 147.

[6] Kennedy, Foster: *American Journal of Medical Sciences*, September, 1919.

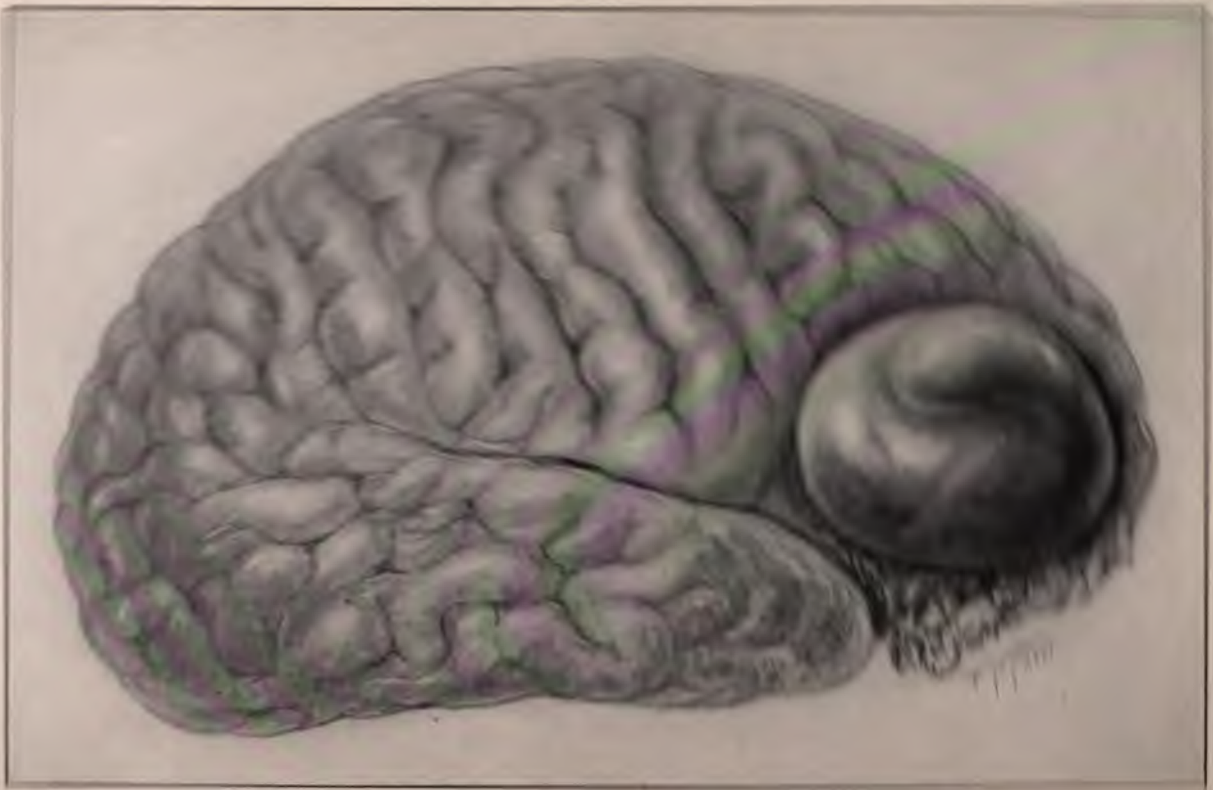

Fig. 40.—Hernia Cerebri. Large hernia following evacuation of frontal lobe abscess from filling of ventricle with pus. No localising symptoms present until sudden paralysis of arms. Hernia probably due to extension into ventricle after operation. (See Fig. 29, page 109.)

(c) *Loss of Abdominal Reflexes of the Same Side.*—Absence or weakness of the homolateral abdominal reflexes probably is frequently present. The author, however, knows of no such observations in abscess, although he frequently has observed it in frontal lobe tumors.

(d) *Sudden Paralysis of Arm of Contralateral Side.*—A pathognomic symptom of frontal lobe abscess is paralysis of the arm of the opposite side to the causative accessory sinus disease. It generally occurs very suddenly; the onset of paralysis from the presence of frontal lobe abscess signifies an abscess of enormous size.

The author has seen a frontal lobe abscess of large size which gave no paralysis whatever—although the abscess had existed for several weeks—until suddenly the arm became totally paralyzed. (Fig. 40.)

(e) *Hemiplegia of Contralateral Side.*—Paralysis of the leg and arm are probably more common in frontal lobe abscess of orbital origin than in cases of frontal sinus origin.

(f) *Mental Disturbances.*—So little is known of the functions of the frontal lobe that it is not surprising that the clinical evidence of intellectual and psychic disturbance should generally be wanting in the reports of frontal lobe abscess.[7, 8, 9, 10]

(g) *Change of Disposition.*—Change of disposition and poor memory may both be present, although they have been but infrequently observed in frontal lobe abscess. In suppuration in the frontal sinus or ethmoids, however, any change in disposition should be regarded as of the greatest significance. The frontal lobes will stand a remarkable degree of compression without any sign of disorientation as to time or place, but in the author's experience one of the most frequent of the changes is in disposition.

In a case of large pial cyst compressing the left frontal lobe the first evidence of its presence was a certain degree of apathy regarding the welfare of the family.

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[7] Bolton, J. S.: *The Brain in Health and Disease; Edward Arnold, London,* 1914, p. 65. Divides the frontal lobe from the functional aspect into posterior, or psychomotor (cerebral-associative-motor), and anterior or prefrontal (higher-cerebral-associative) portions. On page 122 states "the pre-Rolandic portion of the cerebrum or the frontal lobe possesses controlling and executive functions."

[8] Guilliani, G., and Libert, E., observed several cases of extensive destruction of the frontal lobes without disturbance of the psychic functions. Considérations cliniques sur un cas d'abscès du lobe frontal gauche; *Bulletins et Memoirs Société Médicale des Hôpitaux de Paris,* No. 71. p. 680.

[9] Viete, Henry: Notes on War Injuries of the Frontal Lobe; *Albany Medical Annals,* January, 1920, Vol. LXI, No. 1, p. 14. (With extensive destruction of the anterior pole of the left frontal lobe the only symptom was slight incontinence of urine, although he was carefully examined for psychic changes, especially defects in memory, attention, judgment, self-control.

[10] Bing, Robert: Compendium of Regional Diagnosis in Affections of the Brain and Spinal Cord; *New York, Rebman Company,* 1909, p. 202. (Psychic disturbances are observed chiefly in lesions of the anterior portions of the frontal lobe. The lesion must be bilateral, or, if unilateral, it must be capable of producing a sympathetic symmetrical affection of the opposite frontal lobe.")

(2) *Symptoms due to Association between Frontal Lobe and Cerebellum.*—There seems to be an intimate association between the frontal lobe and the cerebellum, as both produce falling. There is some clinical evidence to suggest that in acute lesions spontaneous past-pointing deviations of the contralateral arm also occur.[12]

## ADDITIONAL REFERENCES DIAGNOSIS FRONTAL LOBE ABSCESS.

(a) Keschner, Moses: Large Endothelioma of the Dura Compressing Both Frontal Lobes: *Journal American Medical Association*, June 17, 1916, Vol. LXVI, pp. 1912-1915.

(b) Kennedy, Foster: Retrobulbar Neuritis as an Exact Diagnostic Sign of Certain Tumors and Abscesses in the Frontal Lobes: *American Journal of the Medical Sciences*, September, 1911.

(c) Sierra, A. M.: Nota previa acerca del estudio funcional de los lóbulos frontales; *La Semana Médica*, 1920, Vol. XXVII, p. [illegible].

(d) Milian, G.: Le Coma Frontal. *Paris Médical*, 1921, No. 49, p. 254.

(e) [illegible], L. W.: Brain Abscess as a Complication of Acute Infection of Nasal Accessory Sinus: *Laryngoscope*, 1920, Vol. XXX, No. 3, p. 145.

(f) [illegible], H.: Ueber Stirnhirnabscess: Inaugural Dissertation, 1917; *Jahresbericht für Neurologie und Psychiatrie*, 1917, Bd. XXI, S. 548.

(g) Guillain, G. and Libert, E.: Considérations cliniques sur un cas d'abcès du lobe frontal droit. *Bull. et Mém. Soc. Méd. des Hôpitaux de Paris*, 1920, No. 17, p. [illegible].

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[12] Albrecht, W.: Ueber die Beeinflussung des Bárányschen Zeigeversuchs vom Grosshirn, speziell vom Stirnhirn aus. [illegible] Diseases of the Throat, Nose and Ear [illegible] *Archiv für Ohren-, Nasen- und Kehlkopfheilkunde*, 1920, Bd. [illegible], S. 1.

# CHAPTER XIV.

## COMPLICATIONS AND RESULTS.

**Complications after Evacuation.**—After the evacuation of a chronic brain abscess, if the symptoms continue and meningitis is not present, the surgeon must decide whether the symptoms are caused by a refilling of the abscess or by a suppurative encephalitis. In the former case a further evacuation is called for, while in the latter any irritating manipulation is contra-indicated.

In acute abscess, after evacuation through a small dural opening all symptoms should rapidly subside and not again recur; the paralysis should disappear and the headaches and vomiting cease. Strabismus and papillœdema, however, subside much more slowly; there should be no increase of the latter.

**Differential Diagnosis.**—Encephalitis is distinctly a septic process which is in no way limited, while in a refilled abscess the abscess is partly, if not completely, limited. The differential diagnosis between refilling of the abscess cavity is always difficult, but in a general way a slowly developing compression signifies a refilling of the abscess while exhaustion denotes an encephalitis.

Vomiting after evacuation is a most serious symptom as it usually signifies an extension of the suppurative process.

Headaches may occur either from refilling or extension, but when associated with great prostration they are to be taken as denoting encephalitis.

Irregular pulse is a sign of extension. Progressive paralysis always means extension.

**After Effects.**—No case of brain abscess should be regarded as completely and permanently cured. Cerebral tissue, once the seat of suppuration, never can be regarded as fully normal.

In one of the author's cases of double cerebellar abscess the boy, although he had been perfectly well for many years, when called into service and put under the stress of war developed intense headaches which incapacitated him. In another case, although the small hernia was completely covered and the patient apparently had been perfectly well for several years, rotation in a Barany chair caused a convulsion, the first since the evacuation of the abscess.

Richardson relates a case in which brain abscess had been cured but death occurred suddenly several years later. (Richardson, Charles; Personal communication.)

**Results.**—Contrary to the generally accepted view the mortality of brain abscess is still very high. In the author's cases the mortality following surgical treatment of brain abscess has been about 75 per cent., the recoveries only about 25 per cent. In recent years, since adopting the principle of operating on an early diagnosis, of avoiding during operation all unnecessary trauma with its sequence of secondary œdema, and of providing for the relief of any induced compression, his operative results have greatly improved.[1, 2]

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[1] Mygind reports the results of 207 cases of intracranial disease of various kinds, of otitic origin; forty-two of them of brain abscess, only four of which recovered—or less than 10 per cent. He points out that in all intracranial suppuration the mortality rises to a considerable degree with the onset of any added complication. Of his 207 cases, 35 per cent. had more than one intracranial lesion; of these, fifty cases had two intracranial affections, 24 per cent. of them recovering; ten cases had three pathological processes with 12 per cent. of recoveries, and seven cases had four different intracranial lesions with no recoveries. (Mygind, H.: Otogene multiple intrakranielle Sygdomme; *Ugeskrift for Laeger*, 1920, Vol. 82, No. 22, p. 687.)

[2] Nühsmann states that in all large compilations of otitic brain abscess the percentage of recovery in temporo-sphenoidal lobe abscess is 33⅓ per cent., and in cerebellar about 10½ per cent. (Nühsmann, Th.: Erfahrungen über den otitischen Hirnabscess; *Archiv für Ohren, Nasen und Kehlkopfheilkunde*, 1920, Vol. 106, p. 83.)

The writer is of the opinion that this is somewhat misleading, as all recoveries are apt to be reported, while those which end fatally frequently are not.

# APPENDIX I.

## GUIDE FOR DETAILED NEUROLOGICAL EXAMINATION.

The guide is designed to enable hospital internes or trained attendants, without special training in neurology, to conduct a detailed examination by eliciting and recording the details of all objective and subjective symptoms.

The examination is divided into: (I) General Neurological Symptoms, (II) General Neurological Examination, (III) Cerebral Localization, and (IV) Examination of Cranial Nerves.

Suggested terms with possible interpretations and methods of testing, as well as the normal reaction, are placed in brackets. (*) in the margin indicates examination for unconscious patients. The general sections are indexed by Roman numerals, the chief symptom or manifestations by capital letters, and the individual details by alternate letters and numbers.

## DIRECTIONS FOR USING GUIDE.

The examination begins under the heading *General Symptoms.*

At the completion of the examination all outstanding symptoms and abnormal findings are recorded on the record sheet, followed by the index numbers and letters according to the guide, enabling a ready reference to the exact location of each detail. Example: "Papillœdema, IV., B. 1." When a general symptom has a direct bearing upon the localization, it should be cross-indexed. Example: "Dizziness, I.—d., and IV. f. 2."

## GUIDE FOR NEUROLOGICAL EXAMINATION

(to be followed by House Surgeon)

Full Name ......................................
Address ........................................
Hospital No. .................

*Résumé of History:*

*Résumé of Outstanding Symptoms:*

*Résumé of Abnormal Neurological Findings:*

(Give all Index Numbers and Letters)

..................................................
Signature of House Surgeon.

Date.........................

I. GENERAL SYMPTOMS:

(*a*) *Headache:*

Severity—("mild"; "intense"; "bursting"; "throbbing"; "lacerating").
Location—("frontal"; "temporal"; "behind the eyes"; "pituitary"; "occipital"; "vertex").
In Attacks or Constant.
Initial Appearance; Date of.

(b) *Pain:*
Situation—("nose"; "eyes"; "ears"; "arms"; "legs"; etc.).
Duration.
Initial Appearance.
In Attacks or Constant.

* (c) *Vomiting:*
Frequency.
Type—("projectile" or "regurgitant").
Initial Appearance.

(d) *Dizziness:*
In Attacks or Constant.
Degree—("transient giddiness"; "swaying"; "falling").
Duration.
Initial Appearance.
Description of—("if objects or if patient appears to move").
Duration—("momentary" or "continued").

* (e) *Convulsions:*
General or Localized.
Part Involved.
Associated with Unconsciousness.
Preceded by Aura (Description of Aura: "visual flashes of light"; "objects seen"; "auditory"; "tones or music heard").
During Attack:
Head, to R. to L.
Eyes, to R. to L.
Arms, to R. to L.
Legs, to R. to L.

* (f) *Stupor:*
Initial Appearance.
In Attacks.

(g) *Numbness:*
Situation.
In Attacks.
Initial Appearance.

(h) *Tingling:*
Situation.
In Attacks.
Initial Appearance.

II. GENERAL EXAMINATION:

* (a) *Head:*
1. Wounds: description of.
2. Bleeding from ear; R. L.
3. Bleeding from nose; R. L.
4. Scars: description of.

* (b) *Extremities:*
1. Paralysis of Arm? R. L.
2. Paralysis of Leg? R. L.
3. Anesthesia of Arm? R. L.
4. Anesthesia of Leg? R. L.

- • (c) *Reflexes:*
  - 1. Superficial:
    - a. Epigastric.
    - b. Abdominal.
    - c. Cremasteric.
  - • 2. Deep:
    - a. Knee.
    - b. Ankle.
    - c. Babinski.
    - d. Kernig.
  - • 3. Sphincters:
    - a. Loss of Bladder Control.
    - b. Loss of Bowel Control.
  - 4. Vasomotor:
    - a. Abnormal Flushing?
    - b. Abnormal Sweating?

## III. CEREBRAL LOCALIZATION:

- (a) *Frontal Lobe:*
  - 1. Memory for
    - a. Names—(Father's name; of street where he lives).
    - b. Events.
  - 2. Orientation.
  - 3. Affections.
  - • 4. Aphasia.
    - a. For names or other parts of speech.
    - b. Ability to repeat sentences after examiner.
- (b) *Temporal Lobe:*
  - 1. Aphasia:
    - a. Ability to recognize object and state use, but inability to name. (Knife; key.)
    - b. Ability to recognize proper name and ability to repeat name.
  - 2. Uncinate Gyrus Symptoms:
    - a. Hallucinations of Smell.
    - b. Hallucinations of Hearing.
  - 3. Hemianopsia:
    - a. Right-sided. Left-sided.
    - b. For form.
    - c. For colors.
  - 4. Dreamy state.
- (c) *Parietal Lobe:*
  - 1. Sensory Symptoms:
    - a. Paresthesia; touch, pain, temperature.
    - • b. Anesthesia.
    - c. Astereognosis.
    - d. Muscle sense.
  - • 2. Motor Symptoms:
    - a. Paralysis; where; duration.
    - b. Paresis; where; duration.
    - c. Convulsive Seizures, Sec. I-e. Part involved; Consciousness lost.

- **(d) *Occipital Lobe:***
  - **1. Hemianopsia: right-sided; left-sided.**
  - **2. Visual hallucination: right-sided; left-sided.**
- **(e) *Cerebellum:***
  - **1. Dizziness.**
  - *2. Nystagmus.
  - **3. Romberg.**
  - **4. Disturbances of gait.**
  - **5. Ataxia.**
  - **6. Adiadokokinesia.**

**IV. CRANIAL NERVES:**

- **(a) *First:* ("Olfactory"; Special sense).**
  - **1. Smell ("perfumery") Right nostril.**
    **Left nostril.**
- *(b) *Second:* ("Ophthalmic"; Special sense).
  - **1. Fundus examination.**
  - **2. Vision Right Eye. Left Eye.**
- *(c) *Third; Fourth;* and *Sixth:* (Ocular Movements).
  - *1. Pupils:
    - **a. Equal.**
    - **b. Contracted.**
    - **c. Dilated.**
    - **d. React to light.**
    - **e. React to accommodation.**
  - *2. External Ocular Movements:
    - a. Diplopia. Convergent R. L.
      Divergent R. L.
    - *b. Spontaneous Nystagmus to R. to L.
    - *c. Conjugate Deviation to R. to L.
- (d) *Fifth:* ("Trifacial" sensory and motor).
  - 1. *Sensory Portion:*
    - (a) Anesthesia of
    - (b) Neuralgia of
      - Supra-orbital * (Cornea).
      - Infra-orbital.
      - Inferior Dental.
    - (c) Taste in anterior two-thirds of tongue (sugar; salt).
  - 2. *Motor Portion:*
    - (a) Buccinator.
    - (b) Masseter.
  - 3. *Vasomotor Portion:* (Herpes of brow; of lip).
- *(e) *Seventh:* (Facial; Motor, Sensory).
  - 1. *Motor Portion:*
    - a. Paralysis or Paresis. R. L.
      - Part Involved:
        - Brow.
        - Lower Eyelid.
        - Mouth.
        - Cheek.
        - Palate.
  - 2. *Sensory Portion:* ("Taste in anterior two-thirds of tongue").
  - 3. *Vasomotor:* ("Herpes of Ear").

**(*f*) *Eighth:* ("Auditory" and "Vestibular"; Special Senses).**

1. ***Auditory Portion:***

a. Deafness:

| | | R. ear | L. ear |
|---|---|---|---|
| Hearing | Watch | R. | L. |
| | Whisper | R. | L. |
| | Voice | R. | L. |

(Normal or otherwise)

b. Tinnitus:
(Special auditory tests to be conducted by specialist).
Tuning Forks
Weber Referred to: R. L.
Bone Conduction: Duration of (seconds): R. L.

2. ***Vestibular Portion:***

a. Spontaneous Nystagmus.
b. Spontaneous Vertigo.
c. Spontaneous Pointing Deviation (downward from shoulder)
R. Arm L. Arm
d. RÉSUMÉ OF INDUCED VESTIBULAR REACTIONS (to be filled in by expert after special vestibular tests have been conducted).

1. From turning to Right.
Nystagmus.
Vertigo.
Past Pointing. R. Arm L. Arm.
Falling.

2. From turning to Left.
Nystagmus.
Vertigo.
Past Pointing. R. Arm L. Arm.
Falling.

3. From Douching *Right* Ear
Nystagmus.
Vertigo.
Past Pointing. R. Arm. L. Arm.

4. From Douching *Left* Ear
Nystagmus.
Vertigo.
Past Pointing. R. Arm L. Arm.

SPECIAL VESTIBULAR TESTS (to be conducted by expert).

1. Turning to Right (reactions induced by turning to right, head forward 30 degrees).

a. Induced Nystagmus (normal, horizontal to left).
1. Amplitude.
2. Duration (normal; 26 sec.).
3. Direction (normal, horizontal to left).

b. Induced Vertigo (normal; to left).

c. Induced Past Pointing (shoulder from above; normal; to right).
R. arm to—
L. arm to—

d. Supplementary.
1. Head forward; falling (normal, to right).

- **2. Turning to Left (turning to left, head 30 degrees forward).**
  - **a. Induced Nystagmus.**
    - **1. Amplitude.**
    - **2. Duration (normal; 26 sec.).**
    - **3. Direction (normal; horizontal to right).**
  - **b. Induced Vertigo (normal; to right).**
  - **c. Induced Past Pointing (shoulder from above; normal; to left).**
    - **R. Arm to—**
    - **L. Arm to—**
  - **d. Supplementary.**
    - **Head back 60 degrees.**
    - **Head forward: Falling (normal; to left).**
- **3. Caloric *Right* (reactions induced by douching right ear with cold water, head forward 30 degrees)..**
  - **a. Induced nystagmus (normal; rotary to left).**
    - **1. Amplitude.**
    - **2. Direction.**
    - **3. Duration.**
  - **b. Induced Vertigo (normal; to left).**
  - **c. Past Pointing (shoulder from above; normal; to right).**
    - R. Arm to—
    - L. Arm to—
  - d. Supplementary.
    - 1. Head back 60 degrees.
      - a. Nystagmus (normal, horizontal to left).
      - b. Past Pointing (normal; to right).
        - R. Arm to—
        - L. Arm to—
- 4. Caloric *Left* (reactions induced by douching left ear, head forward 30 degrees).
  - a. Induced Nystagmus (normal, rotary to right).
    - 1. Amplitude.
    - 2. Direction.
    - 3. Duration.
  - b. Induced Vertigo (normal; to right).
  - c. Past Pointing (shoulder from above; normal to left).
    - R. Arm to—
    - L. Arm to—
  - d. Supplementary:
    - 1. Head back 60 degrees.
      - a. Nystagmus (normal; horizontal to right).
      - b. Past Pointing (normal; to left).
        - R. Arm to—
        - L. Arm to—

• (G) *Ninth:* ("Glossopharyngeal"; special sense; sensory motor).
- 1. Taste.
- 2. Swallowing.

• (H) *Tenth:* ("Pneumogastric"; motor and sensory).
- 1. Hoarseness.
- 2. Rapidity of Heart.

(I) *Eleventh:* ("Spinal Accessory"; motor).
  1. Trapezius Muscle.
  2. Sternocleidomastoid.
  3. Shoulder Dropping.

(J) *Twelfth:* ("Hypoglossal"; motor).
  How is tongue protruded?

# APPENDIX II.

## CEREBELLAR ABSCESS: ANALYSIS OF PATHOLOGICAL CONDITION IN REPORTED AUTOPSIES OF 125 CASES.

### *Post-Mortem Findings in*
### *Cerebellar Abscess.*

### Analysis of Cases Reported from 1857 to 1919.

**Dupuy**: Gazette médicale de Paris, XII., 1857, p. 230.

*Condition of Dura Over Site of Infection:* In four cases of cerebellar abscess noted by the author in the literature, the dura mater connected with the cerebellum was more or less altered.

**Gull, W.**: Guy's Hospital Reports, III. Series, 1857, p. 261.

*Situation:* Apparently posterior, but not stated. Stalk. Abscess near cerebellar surface; superficial wall of abscess adherent to dura for a small space corresponding to the entrance of the vein from the vestibular aqueduct. Lateral sinus thrombosis. Hydrocephalus.

*Condition of Dura Over Site of Infection:* Superficial wall of cerebellar abscess was adherent to the dura mater for a small space, corresponding to the entrance of the vein from the vestibular aqueduct.

*Capsule:* The abscess (in the right lobe of the cerebellum near the surface) was limited by "a highly vascular cyst." (This is an old observation dating back to 1857.)

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.)*: The nervous substance surrounding the cerebellar abscess was edematous.

**Heusinger**: Virchow's Archiv, XI., 1857, p. 92.

*Situation:* Double brain abscess, one in cerebellum, one in lower lobe of cerebrum. Situation of cerebellar abscess probably posterior; corresponding transverse sinus entirely obliterated by a solid thrombus. Petrous bone was destroyed in an enormous circumference.

**Schwartze, H.**: Archiv für Ohrenheilkunde, Vol. I., 1864, p. 195.

*Situation:* Apparently anterior, but not stated. Cerebellar abscess originating through passage of pus from mastoid antrum into the internal ear through the oval window and through the internal auditory meatus, into the cranial cavity. Hydrocephalus. No sinus thrombosis.

*Condition of Dura Over Site of Infection:* Dura mater over left petrous bone was very loosely attached and easily removed.

**Voltolini, R.**: Virchow's Archiv, Vol. XXXI., 1864, p. 202.

*Situation:* Probably anterior, but not stated. Cerebellar cortex had undergone local gangrenous destruction; middle portions were greatly softened. Pia mater of cerebellum in part infiltrated with pus. Infection through subarcuate hiatus. Hydrocephalus.

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.)*: The cerebellar cortex had undergone a local gangrenous destruction and the adjacent white substance was softened; the middle portions were very soft. Pia mater of cerebellum was in part infiltrated with pus.

Troeltsch: Archiv für Ohrenheilkunde, Vol. IV., 1869, p. 126.

*Situation:* Apparently posterior, but not stated. Cerebellar abscess and a similar abscess in temporal lobe, applied to petrous bone at tegmen tympani and posterior pyramid, separated by tentorium and by normal brain substance. Labyrinth suppuration. Phlebitis of superior petrosal sinus, in observer's opinion perhaps propagated through the subarcuate hiatus.

*Condition of Dura Over Site of Infection:* Dura mater was discolored, considerably thickened, and covered with superficial fibrinous deposits over the tegmen tympani as well as at the posterior surface of the pyramid, where the brain abscesses (cerebellar and temporal) were applied to the petrous bone.

Gribbon, G. C.: Lancet, I., 1878, p. 712.

*Situation:* "On examining the cerebellum, all of the interior of the organ except about the anterior third of the median and left lobes was found to be an abscess . . . it appeared to have begun in the right lobe, of which nothing was left but the cortex. . . ." Hydrocephalus. (Case of abscess of the cerebellum bursting into the ear with immediate death. No operation.)

*Condition of Dura Over Site of Infection:* The bone was denuded of dura mater and stained with pus for a quarter of an inch around the auditory meatus. Pus also abounded in the mastoid cells.

*Capsule:* The cerebellar abscess appeared to have begun in the right lobe of which nothing was left but the cortex, as here it was encased in a thick lining membrane, while in the other lobes to which it afterwards penetrated there was no such membrane of demarcation, but it was as if the cerebellar substance were being rapidly broken down in every direction.

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.)*: "On slitting up the tentorium, the surface of the cerebellum close to the right auditory meatus was seen to be discolored, and pus to be lying between it and the bone. On examining the cerebellum, all of the interior of the organ except about the anterior third of the median and left lobes was found to be an abscess . . . it appeared to have begun in the right lobe, of which nothing was left but the cortex. It was as if the cerebellar substance were being rapidly broken down in every direction."

Moos, S.–Steinbrugge, H.: Zeitschrift für Ohrenheilkunde, X., 1880-81, p. 87.

*Situation:* Abscess occupied the larger portion of the left cerebellar hemisphere and a small portion of the vermis. Perforation of dura in region of aperture of vestibular aqueduct. Lower end of anterior bony boundary of oval window was carious and necrotic. Carious orifice in promontory corresponding to beginning of first cochlear convolution.

Extensive destruction of left pyramid. Apparently abscess with stalk.

*Condition of Dura Over Site of Infection:* Dura mater appeared thickened and discolored at posterior surface of left petrous bone and presented a small round perforation in the region of the aperture of the vestibular aqueduct.

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.)*: Pia and arachnoid of left cerebellar hemisphere, which contained an abscess were opaque. Dura thickened and discolored, also perforated, at posterior surface of petrous bone.

FINNEL: Transactions New York Pathological Society, IV., 1881, p. 317.

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.)*: Abscess in right hemisphere of cerebellum. Neighboring tissue was much softened.

RAYMOND, F.: Progrès médical, IX., 1881, p. 749.

*Situation:* Cerebellar abscess following otitis media with propagation to internal ear. Abscess occupied the entire left lateral lobe which was completely destroyed. Cavity was well outlined and did not communicate with neighboring regions. Dural adhesions. No special findings in mastoid cells. No remarks concerning hydrocephalus.

*Condition of Dura Over Site of Infection:* Dura mater of normal appearance; no adhesions except at level of left petrous bone, surrounding the internal auditory meatus; in this region the cerebral meninges were thickened and indurated. A portion of the dura mater remained adherent at the level of the abscess around the internal auditory meatus.

*Capsule:* The walls of the cerebellar abscess were thickened, hardened and roughened on the internal surface.

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.)*: The cerebellar abscess occupied the entire left lateral lobe, which was completely destroyed.

HADDEN, W. B.: St. Thomas's Hospital Reports, N. P., Vol. XIV., 1884, p. 297.

*Case I. Situation:* Not stated. Large abscess involving right hemisphere of cerebellum and extending about a third of the way across the left hemisphere. Distinct but not extensive caries of right mastoid cells.

*Condition of Dura Over Site of Infection:* Dura mater at base of skull was healthy; no caries was seen on stripping it.

*Case II. Situation:* Superficial abscess involving under surface of left lobe of cerebellum. Pus in mastoid cells on both sides. History of a fall.

*Condition of Dura Over Site of Infection:* Dura mater and surface of bone at base were healthy.

SCHULZ, R.: Deutsches Archiv für klinische Medizin, Vol. XXXV., 1884, p. 477.

*Situation:* Anterior. Abscess was situated upwards and forwards in the cerebellum. No mention of a stalk. No sinus thrombosis. Hydrocephalus.

*Capsule—(Presence or Absence)*: The right cerebellar hemisphere contained an abscess cavity larger than a walnut, lined with a discolored pyogenic membrane.

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.)*: From the right cerebellar hemisphere, some viscid greenish pus escaped from a small orifice with discolored surroundings, in petrous bone near porus acusticus.

STALLARD, J. H.: Pacific Medical and Surgical Journal, Vol. XXVI., 1884, p. 207.

*Situation:* Traumatic abscess; central part of right lobe of cerebellum softened and reduced to the consistency of cream.

*Capsule:* Central part of right lobe of cerebellum was reduced to the consistency of cream; the softening did not invade the gray matter or approach the surface. On the inner side there was no trace of a limiting membrane but on the outer side there were distinct remains of an attempt at limitation in an oval spot nearly one inch in its longest diameter. This membrane was covered with pus cells. (Case of traumatic origin.)

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.)*: Central part of right lobe of cerebellum was reduced to consistency of cream; this softening did not invade the gray matter or approach the surface.

CAPDEVILLE, F.: Journal de Médecine de Bordeaux, XIV., 1884-85, p. 37.

*Situation:* Abscess sequel of middle ear disease, filled the entire right lobe of cerebellum. No sinus thrombosis; sinuses were entirely intact. Osteitis of petrous bone and mastoid process. Deep cavity filled with pus in petrous bone, at level of angle formed by superior petrosal sinus and right lateral sinus. No mention of hydrocephalus.

*Condition of Dura Over Site of Infection:* The sinuses of the dura mater were entirely intact, free from phlebitis or thrombosis. At the level of the angle formed by the superior petrosal sinus and the right lateral sinus, a deep cavity filled with pus was found in the petrous bone; This cavity was covered by a thinned portion of dura, which however presented absolutely no change or solution of continuity.

*Capsule:* Right cerebellar lobe was filled with greenish pus which was completely enclosed in a continuous shell composed by the cerebellar cortex.

*Pathological Processes in Cerebellum — (Softening, Discoloration, etc.)*: On incision of the right cerebellar lobe, a mass of greenish pus (about 100 grammes) escaped, entirely filling the lobe; this pus was completely enclosed in a continuous shell composed by the cerebellar cortex.

HANFIELD-JONES, M.: Brain (London), VII., 1884-85, p. 398.

*Situation:* Two abscesses close beneath surface, rather nearer the upper than the under part of the cerebellum, separated from each other by a septum of tough brain substance, ½ inch thick. Hydrocephalus. Case probably of syphilitic origin.

*Condition of Dura Over Site of Infection:* (Syphilitic Cerebellar Abscess). Right half of cerebellum was found at autopsy to be firmly adherent to the dura mater of the anterior portion of the posterior fossa, so that the dura was removed together with the cerebellum. Dura mater corresponding to site of the two abscesses was adherent to cerebellum and much thickened by a probably gummatous material.

*Capsule:* The right lobe of the cerebellum contained two abscess cavities lined with a pyogenic membrane, evidently not of very recent date. (Case of Syphilitic origin.)

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.)*: Dura mater corresponding to site of two (syphilitic) cerebellar abscesses was adherent and thickened by gummatous material; pia mater covering the cerebellum was thickened, especially near the site of the abscess.

HEDINGER, A.: Zeitschrift für Ohrenheilkunde, Vol. XIV., 1884-85, p. 56.

*Situation:* Apparently anterior, but not stated. Cerebellar abscess due to propagation of pus from middle ear into cochlea and by means of the vestibular aqueduct into the cerebellum. Caries of petrous bone. No mention of stalk. No mention of sinus thrombosis or hydrocephalus.

*Condition of Dura Over Site of Infection:* No orifice was found in the dura, probably because the small orifice was promptly closed by organized connective tissue after perforation and evacuation of pus to the cerebellum had occurred. The pus in the cerebellum caused extensive destruction and after rupture of the abscess drained below the dura in the direction toward the spinal canal.

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.)*: Pus in cerebellum caused extensive destruction, and after rupture of the abscess drained below the dura in the direction towards the spinal canal.

ROTHHOLZ, H.: Zeitschrift für Ohrenheilkunde, Vol. XIV., 1884-85, p. 184.

*Situation:* Two intercommunicating cerebellar abscesses, the foremost reaching close to the fourth ventricle. Autopsy showed a distinct continuous chain of suppuration from the tympanum along the facial nerve whose bony sheath was defective, into the posterior cranial fossa and from here within the soft cerebral meninges into the cerebellum.

*Capsule:* Two intercommunicating cerebellar abscesses, about the size of a walnut, were enclosed in a thick abscess capsule.

SUTPHEN, T. Y.: Zeitschrift für Ohrenheilkunde, Vol. XVII., 1887, p. 286.

*Situation:* Not clear. Caries of temporal bone corresponding in part to situation of internal auditory meatus; orifice leading from the carious cavity into an abscess in the opposite cerebellum. Adjacent to it above and anteriorly was a mass of cicatricial tissue which is interpreted by the observer as the result of a healed abscess.

*Capsule:* Corresponding to the internal auditory meatus, there was an orifice which led from the large carious cavity in the temporal bone, into an abscess in the opposite cerebellum. Adjacent to the abscess above and anteriorly there was a mass of cicatricial tissue which occupied the entire thickness of the gray matter, was hard to cut, and presented a discolored roughened surface, so that there could be no doubt about its being the result of a healed abscess.

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.)*: Mass of cicatricial tissue, evidently the result of a healed abscess, was found adjacent to a cerebellar abscess above and anteriorly, occupying entire thickness of gray matter.

HUTTON, F. T., and WRIGHT: The Lancet, Vol. II., 1888, p. 210.

*Situation:* Posterior. Cerebellar abscess, in a case of otitis interna, occupying posterior inferior and inner part of left hemisphere. "The petrous portion of the left temporal bone was found extensively diseased, and perforated pus appeared to track backwards between thickened and adherent dura mater beneath the left temporo-sphenoidal lobe towards the cerebellum." No mention of stalk. No mention of sinus thrombosis or hydrocephalus.

*Condition of Dura Over Site of Infection:* Dura was thickened and adherent, and pus passed between it and towards the cerebellum, beneath the left temporo-sphenoidal lobe.

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.):* Some hemorrhage was found at the base of the brain.

SMITH, TH. C.: Journal American Medical Association, Vol. X., 1888, p. 202.

*Situation:* Anterior. "Left hemisphere of cerebellum, anterior half, converted into an abscess." Opening in posterior surface of petrous bone communicated with tympanic cavity. No mention of stalk. No statements concerning hydrocephalus.

*Condition of Dura Over Site of Infection:* Dura mater normal, except on posterior surface of petrous portion of left temporal bone, where the adhesion was a little greater than normal around an opening in the bone, which communicated with the tympanic cavity.

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.):* Anterior half of left cerebellar hemisphere was converted into an abscess.

HARTMANN, A.: Zeitschrift für Ohrenheilkunde, Vol. VIII., 1889, p. 313.

*Situation:* Apparently anterior, but not stated. Cerebellar abscess in a case of otitis media with caries of petrous bone. Total destruction of bony wall of canal of facial nerve at its bend; two carious tracts led from here backwards, one of these tracts corresponding to the hiatus subarcuatus. Abscess with stalk.

REINHARD, and LUDEWIG: Archiv für Ohrenheilkunde, Vol. XXVII., 1889, p. 201.

*Situation:* Anterior. Abscess situated in anterior segment of cerebellum, but there was purulent thrombophlebitis of transverse sinus and superior petrosal sinus. Subdural abscess. Tentorium adherent to cerebellar surface. At the posterior segment of the cerebellum in the region of the upper vermis was a discoloration involving only the pia. Large carious defect in temporal bone; fistulous opening in wall of lower auditory meatus. Apparently abscess with stalk.

*Condition of Dura Over Site of Infection:* The adherent tentorium having been divided, fetid grayish green masses of pus were reached, which spread between the cerebellar surface and the dura.

*Capsule:* Abscess fully the size of a hen's egg, in anterior segment of right cerebellar hemisphere. It was separated from the healthy cerebellar tissue by a green abscess wall, which was almost entirely covered with gangrenous grayish green masses.

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.):* Tentorium cerebelli on right side adherent to surface of cerebellum. Masses of pus spread between cerebellar surface and dura. Large abscess in anterior segment of right cerebellar hemisphere. Surface of cerebellum locally depressed, with irregular orifices in two places from which pus welled up. Grayish yellow discoloration, size of a dime, involving only the pia, at posterior segment of cerebellum in region of upper vermis.

Milligan, W., and Hare, A. W.: British Medical Journal, February 1, 1890, p. 231.

*Situation:* Abscess situated towards middle of right lobe, in its anterior and upper part. No mention of a stalk. Temporo-sphenoidal lobe was normal. No caries of petrous bone. No hydrocephalus. Propagation of infective process is referred by the observers to the small venous and lymphatic channels between the middle ear and the perivascular lymphatic spaces of the cortex.

*Condition of Dura Over Site of Infection:* Membranes and surface of brain were healthy.

*Capsule:* There was an empty and collapsed abscess cavity in the right lobe of the cerebellum, about the size of a small walnut; it was lined by a distinct limiting membrane suggesting that the abscess was of old standing.

Pitt, G. Newton: British Medical Journal, Vol. I., 1890, p. 643.

*Situation:* Abscess situated in upper part of posterior or semilunar lobe of cerebellum; probably secondary to lateral sinus thrombosis. Surface of abscess was adherent to torcular Herophili, where dura and bone were thickened and inflamed. Abscess with stalk. No comments on hydrocephalus.

*Condition of Dura Over Site of Infection:* Dura mater and bone for an inch and a half near the torcular Herophili were thickened and inflamed. (Cerebellar abscess secondary to lateral sinus thrombosis.)

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.)*: Acute softening around pons abscess.

Schwarze: Deutsche Militärärztliche Zeitschrift, Vol. XIX., 1890, p. 535

*Situation:* Case I. Posterior. Abscess in posterior lobe of cerebellum 1 cm. below surface, following otitis media. Evidence of a stalk. Formation of pus focus in cerebellum is explained by observer as the result of phlebitic and periphlebitic processes. Perforation of dura in occipital fossa and partial disintegration of posterior surface of petrous pyramid.

Case II. Situation apparently posterior. Cerebellar abscess due to phlebitic and periphlebitic processes; stump of a blood vessel projected from wall, closed by a plug of pus. No mention of stalk.

*Condition of Dura Over Site of Infection:* Autopsy showed a slight basal meningitis, probably originating from a perforation of the dura, size of a pea, filled with thick pus; dura slightly bulged in the surroundings of the hole and appeared considerably thickened in this region after it had been detached from the skull. Corresponding to the hole in the dura, the occipital fossa also presented a superficial disintegration of the inner bone plate with discoloration.

Panse, R.: Archiv für Ohrenheilkunde, Vol. XXXIII., 1891-92, p. 38.

*Situation:* Apparently anterior. Cerebellar abscess due to infection through vestibular aqueduct. No mention of a stalk. No statement concerning sinus thrombosis or hydrocephalus.

*Condition of Dura Over Site of Infection:* Dura was firmly applied to bone in vicinity of jugular foramen above the transverse sinus.

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.)*: Left cerebellar hemisphere firmly fixed at sharp margin in vicinity of jugular foramen; it contained an abscess fully the size of a pigeon's egg, filled with dirty green pus.

Truckenbrod, C.: Zeitschrift für Ohrenheilkunde, Vol. XXII., 1891-92, p. 228.

*Situation:* Apparently posterior. Cerebellar abscess in a case of osteitis of mastoid process. Transverse sinus thrombosis. Longitudinal sinus was free from thrombosis. Disintegration of dura around cerebellar abscess. No mention of stalk which apparently existed.

*Condition of Dura Over Site of Infection:* Dura was involved in disintegration process around cerebellar abscess.

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.):* Discoloration and softening beyond middle in surroundings of left-sided cerebellar abscess.

Harrison, C. E.: The Lancet, October 1, 1892, p. 763.

*Situation:* Not apparent from description. Cerebellar abscess secondary to ear disease. No mention of stalk. No subdural abscess. No extravasation of pus into meninges or ventricles. No mention of sinus thrombosis or hydrocephalus. Dura over petrous bone discolored and bone necrotic, with small perforation into mastoid antrum, but no evidence of active inflammation in mastoid cells.

*Condition of Dura Over Site of Infection:* Dura mater covering the central part of the anterior surface and to a less extent the posterior surface of the petrous bone, was discolored and under it was a large piece of still undetached necrotic bone.

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.):* Walls of abscess cavity were mostly formed of softened brain tissue.

Randall, B. A.: Transactions American Otological Society, Vol. V., Part I., 1892, p. 93.

*Situation:* Probably anterior, but not stated. Observer considers suppuration to have been limited to the mastoid cells and the adjacent intracranial structures. Autopsy showed an opening from the mastoid beneath the lateral sinuses, communicating with the abscess. No mention of sinus thrombosis or hydrocephalus.

Jansen, A.: Archiv für Ohrenheilkunde, Vol. 35, 1893, p. 272.

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.):* Encephalitic changes in form of hemorrhagic infarction and softening in right cerebellar hemisphere, situated directly below the surface near the temporal bone.

Kenefick, T. A.: Boston Medical and Surgical Journal, Vol. 129, 1893, p. 144.

*Situation:* Cerebellar abscess of apparently spontaneous origin. "Dividing the cerebellum at the base there was found a cavity large enough to hold a billiard ball. The other organs were healthy."

Harbordt: Otitische Erkrankungen des Hirns, etc. (See Körner), 1894, p. 135.

*Situation:* Apparently posterior. Cerebellar abscess following several operations for cholesteatoma of temporal bone. Phlebothrombosis of transverse sinus extending into the jugular vein. Numerous metastatic foci in both cerebral hemispheres, and in other organs.

**Harrison, C. E.:** The Lancet, 1894, II., p. 795.

*Situation:* Apparently posterior. Cerebellar abscess the result of chronic middle-ear disease. No mention of stalk. Sinus thrombosis. Patch of organized lymph on outer wall of lateral sinus, corresponding on its outer surface with a piece of necrotic bone containing holes and depressions through which a probe could in two places be passed into the mastoid cells.

*Condition of Dura Over Site of Infection:* When the dura was separated from the cranium at the autopsy, a patch of organized lymph was found on the outer wall of the right lateral sinus, corresponding with necrotic bone.

**Korner, O.:** Die otitischen Erkrankungen des Hirns, der Hirnhaüte und der Blutleiter; Monograph, 1894, p. 118.

*Situation:* Abscess situated in anterior external portion of cerebellar hemisphere. No mention of stalk. No second abscess was demonstrable anywhere. Cerebellum was adherent to transverse sinus at its knee, and the sinus at the site of adhesion was filled with a thick white partly adherent thrombus; the other sinuses, notably the superior petrosal sinus, were free. No fistula leading to the abscess. Hydrocephalus.

*Condition of Dura Over Site of Infection:* Dura over the petrous bone was normal throughout.

*Capsule:* Abscess cavity size of walnut in anterior external portion of right cerebellar hemisphere; it was lined with a very thin, not everywhere plainly demonstrable membrane.

**Mackenzie, H. W. G.:** St. Thomas's Hospital Reports, Vol. XXII., N. S., 1894, p. 143.

*Situation: Case I.* Chronic middle ear suppuration. Large abscess, almost wholly replacing the left cerebellar lobe. No mention of sinus thrombosis. Hydrocephalus.

*Case II.* Abscess situated in outer third of right cerebellar lobe which was somewhat adherent to outer part of petrous bone; at this spot the dura had disappeared. Old adhesions between medulla and cerebellum. No disease of mastoid cells. The sinuses contained soft non-adherent clot and fluid blood. Hydrocephalus.

*Condition of Dura Over Site of Infection: Case I.* "Dura mater over posterior portion of left petrous bone was blackened and perforated; around the perforation it was adherent to the left cerebellar lobe, which was almost wholly replaced by a large abscess. The bone over the posterior and external portion of the left petrous ridge was found, when the dura was detached, to be carious.

*Case II.* Right cerebellar lobe was somewhat adherent to outer part of petrous bone near its junction with the squamous. At this spot the dura mater had disappeared and there was a small area of bare bone with some loose fragments.

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.): Case I.* Left cerebellar lobe was almost entirely replaced by a large abscess.

*Case II.* Right cerebellar lobe somewhat adherent and enlarged, pale and softened; abscess in outer third of lobe, with well defined walls, separated from surface by fairly normal white matter.

**Wallace, C. S.**: St. Thomas's Hospital Reports, N. S., Vol. XXIV., 1895 (1897), p. 353.

*Situation:* Two cerebellar abscesses (case of mastoiditis), one in anterior part of lateral lobe, at a depth of 1½ inches from cerebellar surface abutting on surface at both ends and forming chord of a circle cutting off the outer and posterior part of the lateral lobe of the cerebellum. Just internal to this and parallel to it was a second abscess cavity, which was elongated, but not so long as the first cavity. No sinus thrombosis. No hydrocephalus.

**Zeller**: Berliner klinische Wochenschrift, 32, 1895, p. 923.

*Situation:* Posterior. Cerebellar abscess in the presence of transverse sinus thrombosis. The site of rupture from the mastoid cavity towards the posterior cranial fossa was situated in the groove of the superior sinus. Fistulas behind the mastoid process, in the parotid region, and in the neck.

*Condition of Dura Over Site of Infection:* The sound could be passed far in between the dura and the bony floor of the posterior cranial fossa.

*Capsule:* Abscess size of walnut in right cerebellar hemisphere, with old pyogenic membrane.

**Woodward, J. U.**: Zeitschrift für Ohrenheilkunde, Vol. XXIX., 1896, p. 373.

*Situation:* Abscess occupied entire left lateral lobe of cerebellum. No lateral sinus thrombosis. Several small orifices in the sinus wall established a communication with pus focus under dura mater. Hydrocephalus. Adjacent cortical portion of cerebellum was discolored, softened and necrotic over a small area.

*Condition of Dura Over Site of Infection:* Purulent pachymeningitis in area on posterior surface of petrous portion of left temporal bone near opening of internal auditory meatus; bone was carious in this area which extended to below the lateral sinus; no lateral sinus thrombosis. Several small orifices in sinus wall communicated with pus-focus under dura mater.

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.):* Cortical portion of left lateral lobe of cerebellum adjacent to abscess was discolored, softened, and necrotic over a small area. Remainder of cerebellar cortex was normal.

**Heimann**: Archiv für Ohrenheilkunde, Vol. 43, 1897, p. 220.

*Situation:* Abscess in right lobe of cerebellum and processus vermiformis, with caries of right petrous bone and general tuberculosis. (The brain abscess was independent of the general tuberculosis and originated at an earlier date, as the remote result of middle-ear suppuration.)

**Müller, R.**: Charité-Annalen, Vol. XXII., 1897, p. 369.

*Situation:* Apparently anterior. Cerebellar abscess through infection of posterior semicircular canal and ductus endolymphaticus. No mention of a stalk, but left cerebellar hemisphere and its dural covering were agglutinated with petrous bone by suppurating tissue strands originating from vestibular aqueduct; caries of petrous bone around aqueduct.

*Condition of Dura Over Site of Infection:* Left cerebellar hemisphere and its dural covering were agglutinated with left petrous bone by suppurating tissue-strands which originated from the vestibular aqueduct;

these strands led from in front towards the pus cavity. Dura was greatly thickened over region of abscess.

*Capsule:* Left cerebellar hemisphere contained a pus focus, size of a walnut. Abscess membrane was in course of formation.

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.)*: Left cerebellar hemisphere and its dural covering were agglutinated with left petrous bone by suppurating tissue-strands derived from vestibular aqueduct.

**Viollet, P.**: Bulletins de la Société Anatomique de Paris, Vol. 72, 1897, p. 450.

*Situation:* Abscess occupied exclusively the central portions of the left lobe of the cerebellum. No sinus phlebitis. No subdural abscess. No mention of stalk. There was a communicating orifice between the middle ear cavities and the cerebellum in front of the sinus, besides granulations in the petrous bone, pointing to this route as the mode of propagation from the ear to the cerebellum.

*Condition of Dura Over Site of Infection:* Communicating orifice between middle ear cavities and cerebellum through the dura in front of the sinus.

**Orne Green, J.**: American Journal of Medical Sciences, Vol. 117, 1899, p. 406.

*Situation: Case I.* Apparently anterior. Caries of labyrinth. Cerebellar abscess, from infection through internal meatus; at base of cerebellum opening against the posterior surface of petrous bone. Adhesion between cerebellar substance and dura between internal meatus and vestibular aqueduct. Abscess was in direct connection with the internal meatus; carious perforation from tympanum to vestibule; mixed infection of brain from vestibule, along internal meatus.

*Case II.* Abscess was situated in the extreme anterior part of the digastric lobe of the cerebellum. It communicated by a fistulous tract with a collection of pus at the orifice of the vestibular aqueduct. Carious perforation from the tympanum to the external semicircular canal, and infection through this to the vestibule and thence along the vestibular aqueduct to the brain.

*Case III.* Abscess in anterior part of digastric lobe of cerebellum: cause, infection through the whole carious osseous labyrinth. Adhesion of cerebellum to dura over vestibular aqueduct. No fistula communicating with the bone was found.

*Condition of Dura Over Site of Infection: Case I.* Small area of cerebellar substance was adherent to the dura, between internal meatus and vestibular aqueduct; no ulceration of dura.

*Case II.* Petrous Bone: Dura was healthy except at vestibular aqueduct, where there was in the bone a collection of pus as large as a bean.

*Case III.* Cerebellum adherent to dura for ¼ inch over vestibular aqueduct.

*Capsule: Case III.* Abscess in anterior part of left digastric lobe of cerebellum, incapsuled by a thin pyogenic membrane.

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.)*: *Case I.* Cerebellum adherent to whole posterior surface of petrous bone; cerebellar convolutions somewhat flattened, probably due to excess of fluid in ventricles.

*Case II.* Slight purulent deposit on upper surface of cerebellum; more abundant deposit on its base on both sides. Pia was inflamed over an area ¾ inch in diameter on right lower surface.

*Case III.* Encephalitis of cerebellum, with abscess of the left digastric lobe from infection through the whole labyrinth.

MUCK: Zeitschrift für Ohrenheilkunde, Vol. 37, 1900, p. 194.

*Situation:* Extradural abscess in posterior cranial fossa. Partial ulceration of saccus endolymphaticus. Orifice led from cavity in inner wall of antrum directly into the subarcuate hiatus. No sinus thrombosis.

OKADA, W.: Klinische Vorträge aus dem Gebiete der Otologie und Pharyngo-Rhinologie, Vol. III., H., 10, 1900, p. 340.

*Position: Case I.* Evidently anterior. Perforation of petrous bone at labyrinth wall, filled with pus. Dura adherent to petrous bone and cerebellum. Cerebellar abscess with punctiform hemorrhages in surroundings. Abscess with stalk. Hydrocephalus.

*Case II.* Abscess situated near anterior circumference of cerebellum which was adherent to the necrotic petrous bone, in surroundings of vestibular aqueduct. Route of infection through oval window. Hydrocephalus. No operation. No mention of stalk which probably existed.

*Case III.* Abscess in cerebellum at the transition of the posterior upper into the anterior lower surface, laterally, where this border is closely applied to the posterior petrous wall directly below the tentorium cerebelli. Suppurating connective tissue strands, coming from the vestibular aqueduct between the internal auditory meatus and the sigmoid sinus, led to the abscess. Necrosis of petrous bone around opening of vestibular aqueduct; the strands led to this point.

*Condition of Dura Over Site of Infection: Case I.* Pachymeningitis circumscripta petrosa, arachnitis adhesiva et purulenta cerebellaris. Dura adherent at anterior and lateral surface of right cerebellum, to petrous bone as well as to cerebellum. Purulent infiltration of dura at this circumscribed spot.

*Case III.* Left cerebellar hemisphere was agglutinated by suppurating tissue strands with the dura covering the posterior surface of the petrous bone; these strands came out from the vestibular aqueduct, between the internal auditory meatus and the sigmoid sinus; they led to a pus cavity in the left cerebellum. After the dura had been stripped from the petrous bone, the bone appeared necrotic around the opening of the vestibular aqueduct, to the extent of a lentil; the suppurating strands led to this point.

*Capsule: Case II.* Abscess barely the size of a walnut was situated near the anterior circumference of the right cerebellum, and lined with a smooth, thick, granulating membrane.

*Case III.* Pus focus, size of walnut, in left cerebellum, at the transition of the posterior upper into the anterior lower surface. A true abscess membrane was not present, but was in course of formation; the abscess was sharply outlined from the surroundings.

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.)*:

*Case I.* Punctiform hemorrhage in surroundings of abscess in right cerebellar hemisphere.

*Case II.* Right cerebellum adherent to petrous portion. Right half of cerebellum œdematous.

*Case III.* Left cerebellar hemisphere agglutinated by suppurating tissue strands with dura covering the posterior surface of the petrous bone.

**Grunert, K.**, and **Zeroni, W.**: Archiv für Ohrenheilkunde, Vol. 51, 1900-01, p. 262.

*Situation:* Apparently anterior. Abscess in right cerebellum. Avenue of infection: fistula of promontory. Labyrinthine suppuration. Vestibular aqueduct was the avenue of infection. Abscess with stalk.

*Condition of Dura Over Site of Infection:* Dura was adherent at posterior surface of petrous portion of temporal bone.

*Capsule:* Abscess in right cerebellar hemisphere, with abscess membrane.

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.)*: Cerebellar substance surrounding abscess was interspersed with extravasates.

**Preysing, H.**: Archiv für Ohrenheilkunde, Vol. 51, 1900-01, p. 262.

*Situation:* Old encapsulated cerebellar abscess, anterior, with stalk. Situation not stated in case of cerebellar abscess. Extradural abscess in immediate vicinity of trephine orifice, with fine perforation of dura; corresponding to this point abscess in cerebellum. Avenue of infection: fistula of dura.

*Condition of Dura Over Site of Infection:* Fistula of dura; abscess in right half of cerebellum, following chronic otitis media and radical operation; extra-dural abscess and fistula close to trephine orifice of right mastoid process.

**Grunert, K.**, and **Schulze, W.**: Archiv für Ohrenheilkunde, LIV., 1901-02, p. 63.

*Situation:* Apparently anterior although the sigmoid sinus contained a thrombus extending as far as the bulb of the jugular vein. Extradural abscess and another abscess at the lower surface of the left cerebellum. The vestibular aqueduct is stated to have been the source of the infection.

*Condition of Dura Over Site of Infection:* Dura was discolored over posterior aspect of middle portion of left petrous bone. Under the dura was a collection of pus and a polyp-like mass which penetrated through the vestibular aqueduct into the internal ear.

**Schulze, W.**: Archiv für Ohrenheilkunde, Vol. LVII., 1902-03, p. 67.

*Situation:* Apparently anterior. Abscess of right cerebellar hemisphere. Avenue of infection: Operative (?) opening of oval window, infection of vestibule and vestibular aqueduct; empyema of saccus; cerebellar abscess by continuity and contiguity.

*Condition of Dura Over Site of Infection:* Cerebellum adherent to dura. Dura greatly thickened at posterior surface of pyramid between sigmoid fossa and porus acusticus internus. On inner surface of dura, at opening of vestibular aqueduct, was a ruptured pus sac.

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.)*: Soft meninges together with cerebellum were agglutinated to abscess wall.

Fliess, H.: Deutsche Medizinische Wochenschrift, No. 14, 1903, p. 242.

*Situation:* Anterior. The abscess was situated at the lower surface of the left cerebellum in the anterior external quadrant. The pus had evidently reached the posterior cranial fossa by way of the vestibular aqueduct.

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.)*: The outer wall of the cerebellar hemisphere which formed a portion of the abscess cavity was entirely destroyed by the suppuration in its anterior half, and this segment was adherent to the petrous bone, being directly agglutinated to the dura, over sac of Cotugno.

von Hinsberg: Deutsche Medizinische Wochenschrift, No. 39, 1904, p. 1245.

*Situation: Case I.* Anterior. No sinus thrombosis or labyrinth suppuration. Abscess in left cerebellar hemisphere could be traced to the antrum through the hiatus subarcuatus, which presented well-marked changes. The superior semi-circular canal—free from necrosis—contained a number of blood vessels imbedded in granulation tissue which was continuous with granulations in the antrum and could be traced to the opening of the hiatus subarcuatus at the posterior edge of the pyramid.

*Case II.* Anterior. Sinus without changes. Abscess in left cerebellar hemisphere was due to infection through the vessels of the hiatus subarcuatus which carried infectious material from the diseased mastoid cells with which they were directly connected, into the dura in the form of emboli.

*Condition of Dura Over Site of Infection:* Cerebellum slightly adherent to pyramidal surface in region of left porus acusticus internus.

von Hinsberg: Dura here presented a thin reddish-yellow deposit and was greatly thickened; centre of dural thickening was situated exactly over opening of hiatus subarcuatus; vessels of canal entered directly into the dura.

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.)*: Under side of left cerebellar hemisphere (which contained the abscess) was covered with purulent deposits.

von Uchermann: Zeitschrift für Ohrenheilkunde, Vol. 46, 1904, p. 303.

*Situation: Case V.* Multiple brain abscess. Cerebellar abscess presumably posterior, in direction of trephining orifice just above knee of thrombotic transverse sinus. Temporal lobe abscess about midway in left middle temporal gyrus.

*Case VII.* Three cerebellar abscesses, apparently posterior; first cavity just inwards of thrombotic transverse sinus; second abscess about in middle of hemisphere; third cavity more mesially situated, in vicinity of but not reaching the median line; extending forwards as far as the flocculus.

Boesch, H.: Zeitschrift für Ohrenheilkunde, Vol. 50, 1905, p. 337.

*Situation:* Anterior. Labyrinth suppuration with right-sided cerebellar abscess. All sinuses were empty. Upper semicircular canal was empty. The anterior and inferior side of the right cerebellar hemisphere presented a discolored focus leading into the abscess cavity which filled the entire portion of the right cerebellum above the arbor vitæ, extending to the left half. In front the cerebellar abscess was directly bounded by the suppurating and necrotic saccus endolymphaticus, plainly showing the path of infection of the brain substance.

*Condition of Dura Over Site of Infection:* Dura was adherent to the bone and to the pia, at site of mastoid operation; pia was adherent to reddish and discolored brain-substance.

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.):* Anterior and inferior side of right cerebellar hemisphere presented a discolored focus, size of a bean, which led into a larger pus cavity; the brain substance in the surroundings was discolored, greenish, interspersed with numerous punctiform hemorrhages.

Politzer, A.: Archiv für Ohrenheilkunde, Vol. 65, 1905, p. 161.

*Situation:* Anterior. Chronic cerebellar abscess in upper vermis of cerebellum. The cochlear capsule presented several irregular gaps bounded by eroded margins, communicating with the cochlear canal, or separated from it only by the endosteum. The process of disintegration undoubtedly originated in the interior of the cochlea. There was also suppuration of the prolapsed temporal lobe, following radical operation for otitis media.

D'Hoore: Archives Internationales de Laryngologie, d'Otologie et de Rhinologie, Tome XXII., 1906, p. 758.

*Situation:* Probably anterior. Sinus apparently normal. Autopsy had to be restricted to an examination through the enlarged bony gap of the mastoid-operation wound. The drainage tube led into an abscess pocket, but beyond this abscess extended another voluminous pouch, tapering at the anterior tip of the right cerebellar lobe. (Stalk?) It was not possible to establish the lesion of continuity between the cerebellar focus and the mastoid across the dura mater.

Knapp, A.: Transactions of the American Otological Society, Vol. IX.-X., 1906, p. 39.

*Situation:* Apparently anterior. Abscess in anterior part of right cerebellar lobe. Destruction by cholesteatoma of most of the external semicircular canal, with involvement of bone directly posterior to canal.

*Condition of Dura Over Site of Infection:* On reflecting the dura on the right side of the convexity of the brain, there was a patch of exudate over the motor area. The bone directly posterior to the external semicircular canal was involved and corresponded to the thickening of the dura, thus showing the path of infection.

*Capsule—(Presence or Absence):* There was no membrane to the cerebellar abscess.

Lannois–Perretière: Archives Internationales de Laryngologie, d'Otologie et de Rhinologie, Tome XXII., 1906, p. 758.

*Situation:* Anterior. Suppurative labyrinthitis with propagation of pus through the internal auditory meatus; discolored area on arachnoid and pia mater of cerebellar fossa directly related on visceral side to a portion of the abscess at the anterior and lateral portion of the right cerebellar hemisphere, above the white central substance. Remainder of hemisphere was œdematous and softened.

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.):* Abscess in right cerebellar hemisphere; covered only by a thin layer of cerebellar cortex. The remainder of the hemisphere was œdematous and softened.

**Black**, W. D.: Laryngoscope, Vol. XVII., 1907, p. 553.

*Situation:* Anterior. Abscess situated near centre of right lateral lobe of cerebellum; it seemed to have no connection with the lateral sinus. The observer believes that the infection occurred through the lymphatics, as the sigmoid sinus and the bony walls around it were healthy.

**Boenninghaus**: Zeitschrift für Ohrenheilkunde, Vol. 54, 1907, p. 245.

*Situation:* Posterior. Sinus thrombosis due to empyema of aberrant pneumatic occipital cell. Autopsy showed a purulent disintegrating thrombus in the sinus at a point corresponding to the situation of the cerebellar abscess.

**Isemer**, F.: Archiv für Ohrenheilkunde, Vol. 74, 1907, p. 244.

*Situation:* Anterior. Cerebellar abscess in medullary substance of right hemisphere, following extensive labyrinth suppuration. Small extra-dural abscess at the posterior pyramidal surface. The existing sigmoid thrombosis was not responsible for the abscesses. According to the autopsy findings, the brain abscess had not originated by continuity from the diseased labyrinth, but the germs were carried into the depth of the cerebellum through retrograde thrombosis and phlebitis of small vessels opening from the brain into the pia-veins; possibly also through the sheath-like lymph spaces.

*Condition of Dura Over Site of Infection:* Cerebellum was adherent to dura, in region of old operation wound (labyrinth-suppuration).

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.)*: Right cerebellar hemisphere was somewhat depressed and on palpation appeared very soft as if fluctuating. Small red foci of softening in surroundings of abscess situated chiefly in the medullary substance, with irregular ragged margins.

**Voss**, F.: Zeitschrift für Ohrenheilkunde, Vol. 53, 1907, p. 57.

*Situation: Case I.* Cerebellar abscess apparently posterior; pus was found at depth of 3 cm. Old healed sinus thrombosis. Second abscess situated at posterior pole of occipital lobe.

*Case II.* Right-sided cerebellar abscess evidently posterior; right-sided transverse sinus thrombosis. Perisinus abscess. Discoloration of adjacent dura. Abscess with stalk.

*Case III.* Small superficial abscess in cerebellum, mesially from sinus. Pus was aspirated from lateral surface of cerebellum at depth of 3 cm. No autopsy.

**Briggs**, G. N.: Lancet, II., 1908, p. 1745.

*Situation:* Anterior. No sinus thrombosis. In the performance of the complete radical mastoid operation, the external semicircular canal was found to be carious, but no fistula could be discovered entering the internal ear through the foramen ovale or foramen rotundum.

**Laurens**, P.: Revue Hebdomadaire de Laryngologie, d'Otologie, et de Rhinologie, Vol. XXIX, 1909, p. 513.

*Situation: Case I.* Anterior. Abscess was found in left lobe of cerebellum, at junction of superior and inferior aspect. Path of infection was evidently either labyrinthine (endo- or peri-lymphatic) or juxta labyrinthine. Abscess with stalk.

*Case II.* Apparently anterior. Old abscess in right lobe of cerebellum, at the level of the upper inner angle of the petrous bone. Sudden rupture into the meninges. Abscess with stalk.

*Condition of Dura Over Site of Infection: Case I.* Area of adhesions between dura and pia mater on posterior surface of left petrous bone; pus escaped from this area in post-mortem removal of cerebellum.

*Case II.* Zone of adhesions between dura and pia on posterior surface of right petrous bone; old cerebellar abscess, of latent development, which opened suddenly into the meninges.

BRAUN, A.: Medical Record, March 26, 1910, p. 535.

*Situation:* Probably posterior. Sinus thrombosis. Abscess was found on the surface of the right lobe of the cerebellum, a short distance behind the obliterated sinus. Observer does not think it likely that the cerebellum became infected after the mastoid operation by extension from the infected sinus; but he comments that the cerebellar abscess may have been present at the time of the mastoid operation. There was no necrotic bone.

FLATAU: Monatschrift für Ohrenheilkunde, 41, 1910, p. 910.

*Situation:* Posterior. Cerebellar abscess by way of sphenoid sinus which contained another abscess.

UFFENORDE, W.: Beiträge zur Anatomie, Physiologie, Pathologie und Therapie des Ohres, der Nase and des Halses, Vol. III., 1910, p. 86.

*Situation: Case I.* Apparently posterior. Wall of right sigmoid sinus was covered with a layer of pus. Defect in tegmen tympani. Purulent exudate and dark red hemorrhages at lower aspect of cerebellum, extending in part also to the upper cortical layers.

*Case II.* Probably anterior. Empyema of labyrinth; saccus empyema; rupture of saccus. Cerebellar floor was covered with a smeary layer of pus.

*Condition of Dura Over Site of Infection:* Dura in posterior portion of right middle cranial fossa and in anterior portion of right posterior cranial fossa was covered with a fibrinous layer. Inner parietal layer of dura was smooth and shiny.

***Pathological Processes in Cerebellum—(Softening, Discoloration, etc.):***

*Case I.* Lower aspect of cerebellum showed some purulent exudate and dark red hemorrhages extending in part also to the upper cortical layers.

*Case II.* Cerebellar floor was covered with a smeary purulent layer. Pus in depth of tissues.

BARR, J. STODDART: Glasgow Medical Journal, Vol. 76, 1911, p. 50.

*Situation:* Posterior. Infective paralabyrinthitis and septic thrombosis of the lateral sinus; opening in sinus wall leading into abscess cavity. (No mention of stalk.)

DE STELLA: Annales des Maladies de l'Oreille et du Larynx, Vol. XXXVII., 1911, p. 999.

*Situation:* Probably anterior. Abscess in the vermis and in a large portion of the right cerebellar hemisphere; following radical operation. No details.

DORTES, P.: Revue Hebdom. de Laryngologie, d'Otologie, et de Rhinologie, Tome XXXI., 1911, p. 145.

*Situation:* Anterior. Distinctly outlined abscess in anterior half of left lobe of cerebellum at level of anterior third. Chronic pyolabyrinthitis complicating chronic middle ear suppuration. Osteitis of inner wall and threshold of aditus. Greenish discoloration of posterior surface of petrous bone behind internal auditory orifice. Slit-shaped fistula of external semicircular canal.

*Condition of Dura Over Site of Infection:* At level of discolored area on posterior surface of petrous bone, behind internal auditory orifice, some pus was found under the dura and around the internal auditory orifice.

***Pathological Processes in Cerebellum—(Softening, Discoloration, etc.):*** Left lobe of cerebellum at level of anterior third, presented a black necrotic spot.

Jacques, P., and Coulet, G.: Le Larynx, IV., 1911, p. 50.

*Situation:* Anterior. Large abscess in anterior portion near inferior surface of right lobe of cerebellum, at point corresponding to thrombotic lateral sinus. Focus of pachymeningitis at level of sinus thrombosis. Small area of osteitis in petrous bone near floor of antrum. (No mention of stalk.)

*Condition of Dura Over Site of Infection:* Focus of pachymeningitis at level of a sinus thrombosis.

***Pathological Processes in Cerebellum—(Softening, Discoloration, etc.):*** Inferior surface of right lobe of cerebellum presented a greenish purulent spot at a point corresponding to the lateral sinus; the cerebellar cortex underneath this spot was softened and peculiarly thinned, but not ulcerated. Wide area of purulent infiltration in the form of a greenish areola, around abscess cavity.

Kelson, W. H.: Proceedings of the Royal Society of Medicine: Otological Section, Vol. IV., Part III., 1911, p. 40.

*Situation:* "The brain shows the site of the abscess surrounded by œdema and a small lesion (abscess?) on the dorsal surface of the left hemisphere of the cerebellum." (No details. Patient died from cerebellar abscess following operation (for nasal polyps) and otitis media).

Logan Turner, A., and Reynolds, F. E.: Journal of Laryngology, Rhinology, and Otology, Vol. XXVI., No. 2, 1911, p. 57.

*Situation:* Posterior. Large abscess in left lobe of cerebellum. Sigmoid sinus thrombosis. No mention of stalk. No meningitis.

Beck, K.: Zeitschrift für Ohrenheilkunde, Vol. 64, 1911-12, p. 262.

*Situation:* Probably anterior. Microscopical specimens plainly showed the route of the infection from the original focus of suppuration in the middle ear to the dura, the cerebellum, the abscess itself, and the scattered encephalitic foci. Extradural abscess due to rupture of mastoid suppuration into extradural space. The encephalitic foci were shown by the specimens to have originated, not by continuity, but through retrograde distribution of the infectious agents by preformed channels.

*Condition of Dura Over Site of Infection:* Strong connective tissue strands passing from the dura formed part of an enormous connective tissue capsule of an old abscess-cavity. Dura was entirely infiltrated with pus; pia and arachnoid were indistinguishable.

*Capsule:* In a case of healed cerebellar abscess, the autopsy showed strong connective tissue strands passing from the dura and forming part of an enormous connective-tissue capsule of the old abscess cavity.

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.)*: A very thin layer of greatly altered cerebellar substance was seen between the agglutinated hard and soft meninges and the cerebellar abscess.

Karsner, H. T.: Journal of Medical Research, Vol. XXV., 1911-1912, p. 393.

*Situation:* Large abscess in middle lobe of cerebellum, extending slightly and equally into both lateral lobes. Probably of metastatic origin.

*Capsule:* The middle lobe of the cerebellum was almost completely replaced by a non-encapsulated deeply seated abscess which extended slightly and equally into both lateral lobes.

Brock, W.: Zeitschrift für Ohrenheilkunde, Vol. 66, 1912, p. 267.

*Situation: Case VI.* Apparently anterior. Abscess in right cerebellar hemisphere following labyrinth suppuration; pus ruptured from the basal convolution into the internal auditory meatus. Cochlear aqueduct was filled with pus in its entire extent, as was also the vestibular aqueduct in its first portion.

Forgue, E., and Rauzier, G.: La Provence Médicale, No. 43, 1912, p. 481. also

Rauzier and Roger: Montpellier Médical, XXXV., 1912, p. 134.

*Situation:* Posterior. The suppuration starting from the left mastoid passed through the postero-superior aspect of the petrous bone in its perisinusal portion, in front of the permeable lateral sinus, causing a certain degree of meningitis and invading the left lobe of the cerebellum where the pus collected in the form of a small superficial abscess.

*Condition of Dura Over Site of Infection:* Considerable thickening of cranial meninges at level of postero-superior aspect of petrous bone.

Glegg, W.: British Medical Journal, I., 1912, p. 608.

*Situation:* Probably anterior. Abscess in middle lobe of cerebellum; another temporo-sphenoidal lobe abscess, immediately adjacent to cerebellar abscess; tentorium between was intact. (Death some weeks after radical mastoid operation; no details.)

Linck: Zeitschrift für Ohrenheilkunde, Vol. 65, 1912, p. 206.

*Situation: Case II.* Anterior. Abscess due to transition of labyrinth suppuration to cerebellum through ductus and saccus endolymphaticus, by way of a fistula in the bony labyrinth capsule. The primary entrance avenue was the horizontal semicircular canal, the infection travelling backwards through the vestibular aqueduct. Rarefying osteitis of petrous bone.

*Condition of Dura Over Site of Infection:* Discolored yellowish area size of a pea, with irregular margins, on dural covering, corresponding to saccus endolymphaticus.

Savariaud: Journal de Médecine de Paris, No. 9, 1912, p. 171.

*Situation:* Probably anterior. (No sinus thrombosis.) Right lobe of cerebellum was almost entirely occupied by a large abscess without true walls. Section of petrous bone near upper posterior border showed a large fistulous tract filled with granulations and communicating with the tympanum.

*Condition of Dura Over Site of Infection:* Dura m thickened and when incised showed the arachnoid cavity to be filled with pus, especially on right side at base, at the level of the cerebellar fossa.

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.)*: Right lobe of cerebellum was almost entirely occupied by an abscess which had no true walls.

Spencer, F. R.: Annals of Otology, Rhinology and Laryngology, Vol. XXI., 1912, p. 321.

*Situation:* Cerebellar abscess of otitic origin. Floor of abscess 3 to 5 cm. above inferior surface of left hemisphere, which was symmetrically enlarged as a whole. Lobus biventer and lobus gracilis were apparently entirely replaced by the abscess cavity and its limiting membranes. No connection of the abscess could be traced with the sinuses, the mastoid bone, or the inner ear. The process was presumably embolic in character.

*Capsule:* The abscess cavity was surrounded by a clearly defined pyogenic membrane, one-half millimetre in thickness.

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.)*: Inferior surface of left cerebellar hemisphere which was symmetrically enlarged, showed, in the sulci, streaks of yellow purulent material shining through the œdematous pia.

Uchermann, V.: Zeitschrift für Ohrenheilkunde, Vol. 66, 1912, p. 49.

*Situation:* Anterior. Cerebellar abscess originating from petrous portion of temporal bone, namely, the paralabyrinthine cells, (paralabyrinthitis) without a sign of labyrinth suppuration.

Urbantschitsch, E.: Monatschrift für Ohrenheilkunde, Vol. 46, 1912, p. 36.

*Situation:* Anterior. Abscess mesially situated in upper vermis extending symmetrically to both cerebellar hemispheres. Recent thrombosis of left sigmoid sinus. Fistula of semicircular canal and labyrinth-suppuration.

*Capsule:* Abscess size of walnut in upper vermis extending symmetrically to both cerebellar hemispheres, with a not very thick pyogenic membrane.

Bruhl: Berliner Klinische Wochenschrift, No. 19, 1913, p. 897.

*Situation:* Cerebellar abscess in lobus biventer at a distance of 3 cm. from the surface. No mention of a stalk.

Hibler, E.: Centralblatt für Bakteriologie, Vol. 68, 1913, Abt. I., p. 257.

*Situation:* Posterior. Case of cerebellar abscess due to anaerobic infection, in the presence of chronic middle ear suppuration, sinus thrombosis and carcinosis of right petrous bone. Propagation of infection from middle ear to cerebellum presumably occurred by way of and on the soil of the carcinoma which had proliferated into the dural covering of the petrous bone only at this point, where the dura was badly infiltrated and congested. This small area was situated directly over the sigmoid sinus, where the dura of the posterior petrous surface was changed and destroyed.

*Condition of Dura Over Site of Infection:* Dura of posterior petrous pyramid was destroyed in a small area just above the sigmoid sinus. (Proliferation of carcinoma into dural covering of petrous bone.) In a large area of the posterior and in part also the upper surface of the petrous bone, dura was infiltrated, thickened and discolored.

*Capsule:* There was no trace of a pyogenic membrane (right sided cerebellar abscess).

Michaelsen, U.: Zeitschrift für Ohrenheilkunde, Vol. 67, 1913, p. 262.

*Situation: Case VIII.* Anterior. No statement is given as to the site, but the cerebellar abscess had existed six months, so its location must be anterior. Abscess with capsule. Internal hydrocephalus. Fresh thrombosis in sigmoid sinus. Circumscribed labyrinth suppuration in vestibule and semicircular canals, extending through endolymphatic duct, causing saccus empyema and cerebellar abscess.

*Case IX.* Probably anterior. Middle ear suppuration and involvement of labyrinth. Cerebellar abscess derived from saccus. No mention of stalk.

*Case XII.* Probably anterior. Route of infection, labyrinth; dura and pia adherent. Cerebellar abscess; no statement as to stalk. Hydrocephalus.

*Case XIII.* Undecided. No statement as to site of cerebellar abscess or paths of infection. Hydrocephalus.

*Condition of Dura Over Site of Infection:*

*Case XII.* Suppuration in left labyrinth extending to dura of posterior cranial fossa and pia of cerebellum.

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.):*

*Case IX.* Encephalitis, especially of cerebellum, probably derived from saccus.

*Case XII.* Labyrinth suppuration extended to pia of cerebellum.

Maier, M.: Archiv für Ohrenheilkunde, Vol. 95, 1914, p. 163.

*Situation: Case III.* Probably anterior. (Apparently well forward, but not stated.) Abscess with stalk communicating with porus acusticus internus. Serous meningitis. Should have been explored from in front because of the sequestra and the stalk to porus acusticus internus.

*Case IV.* Probably anterior. No sinus thrombosis. Abscess with stalk; from description must have gravitated downward toward floor of posterior fossa.

*Case VII.* Cerebellar abscess; no stalk. Cannot tell location; partial sinus thrombosis. Perisinus abscess.

*Case XI.* Cerebellar abscess with stalk. Cannot state location. Transverse sinus thrombosis.

*Case XIII.* "Lateral margin of cerebellum." Right-sided abscess with stalk 1½ cm. deep. No sinus thrombosis. Cholesteatoma of petrous bone (left). Also apparently an intrapial abscess by extension going to right side.

*Case XIV.* Labyrinth fistula, posterior upper portion of horizontal semicircular canal. Situation of cerebellar abscess apparently lateral and anterior. No stalk. (Funnel-shaped defect in lower surface of cerebellar hemisphere may be stalk.) Hydrocephalus. Fresh clot in transverse sinus and adhesion of sinus to cerebellum. Cannot say where abscess was really situated or trace mode of invasion. The thrombus may have been the cause by adhesive process.

*Case XIX.* Otitis interna purulenta (left). Abscess in left cerebellar hemisphere. Intrapial abscess of right side (subdural space).

No stalk. No description, no location. Cause: circumscribed labyrinthitis.

*Case XXI.* Abscess with a stalk, with necrosis of dura. No sinus thrombosis. Situation, lower surface of cerebellum.

*Case XXVI.* Posterior. Sigmoid sinus thrombosis. Abscess situated at posterior lower surface of cerebellum (behind). No stalk.

*Case XXXI.* Probably anterior. No route given. Right cerebellar hemisphere was enlarged and compressed the pons and medulla, so possibly anterior. No statement as to stalk or exact position.

*Case XXXIV.* Sinus thrombosis. Situation of abscess, lateral half of cerebellar hemisphere. "Abscess had evidently originated from surface." Must have come from sinus thrombosis. Extradural adhesion of posterior and upper surface of petrous pyramid. Abscess with stalk.

*Case XXXVI.* Anterior. Sinus thrombosis. Cerebellar abscess; no statement as to stalk; infection passed probably from sinus to brain surface. Situation "at lateral margin of lower segment," so must be "forward" low down towards bulb.

*Condition of Dura Over Sites of Infection:*

*Case VII.* Inner surface of tense dura mater covered with a layer of thick pus; likewise arachnoid.

*Case XI.* Margins of left cerebellar abscess cavity.

*Case XVII.* Transverse sinus passed in an adhesion between dura and cerebellum.

*Case XXI.* Dura of left cerebellum was transformed into a thickened tissue mass adherent to pia.

*Case XXXIV.* Pachymeninx raised from bone by purulent exudate, at posterior and upper surface of petrous pyramid.

*Case XXXVI.* Thick layer of pus between arachnoid and pachymeninx of left cerebral hemisphere. Also pus in subdural space at the base, especially in the left and right posterior cranial fossa. (Abscess at lateral margin of lower segment of right cerebellar hemisphere.)

*Capsule:*

*Case III.* Large abscess in right cerebellar hemisphere, with pyogenic membrane.

*Case IV.* A continuous membrane could be detached only from the outer half of the abscess cavity in the right-cerebellar hemisphere.

*Case XIII.* Abscess in left cerebellum. Under tentorium on both sides, there was a purulent membrane firmly applied to tentorium throughout; the two cerebellar hemispheres were likewise covered by a fairly adherent purulent membrane.

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.):*

*Case IV.* Right cerebellar hemisphere adherent at its lower surface to petrous bone.

*Case VII.* Cerebellum greatly softened in surroundings of abscess cavity.

*Case XI.* Cerebellar substance around abscess slightly discolored.

*Case XIII.* Cerebellar substance surrounding abscess cavity was greatly softened.

*Case XIV.* Right cerebellar hemisphere very congested and reddened, especially in its lateral areas. Large funnel-shaped defect with reddened margins on lower surface of this hemisphere.

*Case XIX.* Pia mater at base of cerebellum infiltrated with pus. Left cerebellar hemisphere consisted of a disintegrating mass and contained a large cavity.

*Case XXI.* Dura of left cerebellum was transformed into a thickened tissue-mass adherent to the pia. Lower surface of cerebellum presented an orifice which led into a cavity from which the pus had been emptied in an operation.

*Case XXXI.* Right cerebellar hemisphere was enlarged and compressed the pons and medulla.

*Case XXXIV.* At base of brain on lower side of right cerebellar hemisphere, the internal meninges were purulent and infiltrated. Abscess had originated from surface.

**Ruttin, E.:** Monatschrift für Ohrenheilkunde, Vol. 48, 1914, p. 728.

*Situation:* Probably posterior. Sinus thrombosis. Cerebellar abscess; no statement as to situation. No statement as to stalk.

**Friesner, I.:** Annals of Otology, Rhinology and Laryngology, Vol. XXVI., No. 3, 1917, p. 847.

*Situation:* Anterior. Abscess was entered from Trautmann's triangle, "downward and backward" one inch. Sinuses normal.

***Condition of Dura Over Site of Infection:*** Dura macroscopically normal; no granulations, no exudate.

**Syme, W. S.:** Journal of Laryngology, Rhinology and Otology, Vol. XXXII., 1917, p. 190.

*Situation:* Posterior. The deep wall of the lateral sinus was bulging and fluctuating; it was incised and a large cerebellar abscess was opened into. Infection presumably occurred through the sinus and the communicating veins.

*Pathological Processes in Cerebellum—(Softening, Discoloration, etc.)*: Destruction of cerebellum was very extensive and was probably caused by infection from several sources.

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# APPENDIX III

## *FRONTAL LOBE ABSCESS*

### Analysis of 140 Reported Cases.

Total Number Frontal Lobe Abscess, 140.[1]

| | | |
|---|---|---|
| I—Adjacent Frontal Lobe Abscess | | 104 |
| From Frontal Sinus | 64 | |
| From Ethmoid | 23 | |
| With Orbital Complications | 41 | |
| Associated with Osteomyelitis | 9 | |
| II—Metastatic Frontal Lobe Abscess | | 14 |
| In Opposite Lobe | 7 | |
| Of Aural Origin | 9 | |
| III—Traumatic Frontal Lobe Abscess | | 22 |
| | | 140 |

Additional Tabulations Frontal Lobe Abscess.

| | |
|---|---|
| I—Path of Infection of Frontal Lobe Abscess | 71 |
| II—Frontal Lobe Abscess Associated with Complications | 117 |
| III—Frontal Lobe Abscess Associated with Epidural Abscess | 3 |
| IV—Frontal Lobe Abscess Associated with Extradural Abscess | 14 |
| V—Frontal Lobe Abscess Associated with Subdural Abscess | 4 |
| VI—Frontal Abscesses Associated with Meningitis | 31 |

### ADJACENT FRONTAL LOBE ABSCESS.

PREYSING: (1) Left frontal abscess following empyema of left maxillary sinus frontal sinus, and ethmoid cells.

REINHARD: (1) Left frontal abscess with an extradural abscess.

SOLOWYKCZ and KARBOWSKI, B.: (1) Chr. latent frontal sinus suppuration with epidural abscess at greater wing of sphenoid bone.

EAGLETON: (1) Case XXI—D. McS., Chapter Seven, p. 111. Orbital and extradural.

MANASSE: (1) Intracranial complications after acute influenza. Sinusitis. First abscess in left frontal lobe. Then abscess in left temporal lobe and intrameningeal abscess in left occipital lobe.

ZEMANN: (1) Multiple abscesses in rt. frontal lobe, one superficial cortical and two communicating deep seated.

PICQUÉ: (1) Intracerebral abscess after right mastoid symptoms and submeningeal symptoms supervening on influenza.

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[1] The cases in the following lists of Frontal Lobe Abscesses necessarily overlap to a greater or less extent, each list containing many duplicate cases from other lists, according as the various symptoms complicate one with another of the different manifestations. If this is borne in mind there will be no confusion in regard to the 140 recorded cases here given.

**Gruening**: (1) Chronic suppuration ethmoid cells and frontal sinus; osteotomy for exploration of brain; inner plate orbital process removed, dura mater exposed; it was discolored and perforated on under surface frontal lobe. Death.

**Preysing**: (1) Left-sided frontal abscess from gangrenous purulent process in left nose and its cavities.

**Steese**: (1) Abscess in left frontal lobe. Post-mortem showed frontal sinus disease.

**Koebel**: (1) Abscess right frontal lobe after mastoid, antrum and frontal sinus suppuration rt. side. Abscess referred to ear disease. Nasal condition caused no disturbance. Enlarged right frontal sinus and left to a less extent filled with pus. Dura detached and ulcerated.

**Luc**: (1) Rt. frontal abscess after bilateral chr. frontal sinus suppuration.

**Rafin**: (1) Rt.-sided frontal sinusitis and brain abscess.

**Treitel**: (1) Abscess left frontal lobe following removal of polyps left nose, and purulent discharge left nose, and pain in forehead and cheek.

**Silex**: (1) Abscess both frontal lobes after empyema ethmoid cells and frontal sinus.

**von Schröder**: (1) Abscess rt. frontal lobe after rt.-sided retrobulbar phlegmon.

**Krecke**: (1) Left frontal abscess after nasal discharge pus, polyps, and swelling over left eye. Frontal sinus filled with pus.

**Redtenbacher**: (1) Abscess of eyelid after influenza followed by pus in frontal sinus with necrosis and abscess at apex of frontal lobe.

**Williamson**: (1) Abscess anterior part frontal lobe after influenza with profuse nasal discharge and frontal and occipital headache.

**Weichselbaum**: (1) Abscess rt. upper eyelid. Pus in rt. maxillary and frontal sinus and between posterior wall of rt. frontal sinus and dura mater. Pachymeningitis and leptomeningitis and brain abscess.

**Sillar**: (1) Abscess frontal lobe and empyema left frontal sinus after long standing sinusitis.

**Renton**: (1) Abscess rt. frontal lobe after obtaining pus in incision of right upper eyelid for orbital cellulitis. Right orbital plate necrosed. No communication between pus from orbit and cerebral abscess.

**Reiner**: (1) Abscess left frontal lobe with cerebral symptoms and bone disease for 2½ months. Vomiting, fever, convulsions. Extensive caries frontal bone. Meningitis.

**Bousquet**: (1) Rt. frontal abscess. After acute empyema of right frontal sinus, subperiosteal orbital abscess and necrosis of frontal bone. Trephining of frontal bone some hours before death.

**Jacubasch**: (1) Large abscess frontal lobe. Second abscess further back. Previously sore in nose. Dull headache, vertigo, vomiting.

**Maas, H.**: (1) Nasal polypi and nasopharyngeal polyp removed, followed by apathy and vertigo, exophthalmos, delirium, and death. Post-mortem showed dura base of skull covered with pus. Abscess left frontal lobe of brain above ethmoidal region.

**Youl**: (1) Traumatic abscess opposite wound in temporal region. (Frontal lobe?) Abscess communicated with lateral ventricle. No cerebral symptoms until two weeks after injury when child vomited and later had convulsions terminating in death.

**Symonds, C. J.**: (1) Chr. suppuration of ethmoid sinus. Death from diffuse meningitis. Post-mortem showed two abscesses under surface left frontal lobe. Dura mater beneath frontal lobe adherent to brain. Neither abscess opened lateral ventricle. Extension to orbit.

**Symonds, C. J. (second case)**: (1) Chr. suppuration ethmoidal and frontal sinuses with abscess left frontal lobe. Influenza. Nasal discharge. Operation for suspected subdural or cerebral collection. Death. Cerebral infection from ethmoid sinus. Extension to orbit.

**Schaefer, H.:** (1) Orbital phlegmon with death from meningitis. Post-mortem showed abscess. Destruction rt. ethmoid labyrinth rupturing through lamina papyracea resulting in osteitis and periostitis of orbit. Later pachymeningitis and purulent leptomeningitis. Death.

**Struycken, H. J. L.:** (1) Severe headache; pus from right nostril. Frontal sinus opened. Cerebral symptoms. Puncture cranial cavity. Drainage. Recurrence brain symptoms. Later a second abscess opened. Incomplete recovery with persistent amnesia.

**Struycken, H. J. L.** (second case): (1) Right-sided headache with rapidly developing œdema of surroundings of rt. eye and protrusion of eyeball. Ethmoid cells opened and most of them removed. Large abscess behind eyeball opened. Later rigidity nape of neck. Vomiting. Later another operation and another abscess found. Recovery with partial amnesia and persistent loss of smell rt. side.

**Manasse:** (1) Acute accessory sinus suppuration which led to enormous orbital phlegmon. Frontal sinus revealed pus during after treatment. Cerebral symptoms in form of vomiting, headaches and fever. Frontal sinus diseased and closed ethmoid cell found attached to its posterior wall. Three operations, and during last frontal lobe brain abscess found under this frontal ethmoid cell. Suppurative process passed from frontal ethmoid cell to bone and then to cranial contents through preformed vascular channels.

**Uffenorde, W.:** (1) Ozena and left-sided frontal sinus and ethmoid suppuration leading to orbital phlegmon through fistula. Operations (2) revealed extradural and frontal sinus abscess. Two months later frontal abscess; meningitis; after rupture of frontal lobe abscess into ventricle. Death.

**Wiener:** (1) Persistent headache; empyema frontal sinus right side with two operations. Later frontal lobe abscess. Operated. Recovery.

**Hosch, P. H.:** (1) Bilateral maxillary sinusitis and right-sided frontal and ethmoidal sinusitis. Extradural perisinus abscess and frontal lobe abscess found at base of right frontal brain, only after death, because of unusual location. Purulent meningitis.

**Maffei:** (1) Partial loss of vision followed by vomiting and severe headache. Incipient neuroretinitis. Later pain in nape of neck and rise of temperature. Death from asphyxia. Spontaneous discharge from left nostril and filling throat. No operation. No autopsy, but author believed disease to be in frontal lobe.

**Meurers:** (1) Bilateral frontal sinusitis with abscess rt. frontal lobe. Sudden pain left eye. Operation left frontal sinus. Recurrence severe headache and sudden death under collapse. Post-mortem showed abscess rt. frontal lobe; delicate membrane. Cranial sinuses free from pathological changes.

**Heiman, Th.:** (1) Bilateral empyema antrum of Highmore and left-sided frontal sinusitis. Bilateral earache. Severe headache and vomiting. Sudden death. Autopsy showed extradural abscess. Right lobe and both frontal sinuses, ethmoid and maxillary sinus filled with pus. Abscess with stalk, diffuse meningitis.

**Grünwald, L.:** (1) Ozena Empyema both frontal sinuses. Carious destruction posterior sinus wall. Right-sided pachymeningitis. Abscess frontal lobe discovered eight days after primary operation. Recovery.

**Mullin, W. V.:** (1) Pain over frontal sinuses and pus from nasofrontal ducts. Later pus from frontal sinuses and pain across forehead, more severe left side. Sudden death. Autopsy showed abscess left frontal lobe. Necrosis extended from surface all way through frontal lobe and perforated into left lateral ventricle.

**Berens, T. P.:** (1) Pansinusitis rt. side. Radical external frontal ethmoidal and sphenoidal operation performed. Gradual recovery. Three years later frontal headache. Swelling of cicatrix rt. frontal region. Incision through old frontal wound revealed frontal lobe abscess. Author is convinced patient carried large collection of pus for months in frontal lobe, and that this was source of nasal trouble.

**Piffl, O.**: (1) Chr. left-sided frontal sinusitis and exophthalmos. Radical operation. Removal orbital roof and incision of exposed dura. Frontal lobe adherent to orbital roof and frontal abscess found at autopsy.

**Elschnig, A.**: (1) Chr. tuberculous tumor lachrymal gland right eye. Operation. Extirpation of tumor and temporary resection of lateral orbital wall. Death four weeks after. Abscess found in right frontal lobe directly above base with circumscribed adhesions of meninges.

**Elschnig, A.** (second case): (1) Phlegmon left upper eyelid incised. Paresis right side and mental apathy. Operation left frontal abscess. Removal orbital roof and dura. Recovery.

**Elschnig, A.** (third case): (1) Chr. frontal sinusitis. Incipient orbital phlegmon. Radical operation after incision at upper inner orbital margin. Abscess found in frontal brain; death. Autopsy showed abscess reaching to area of second frontal convolution. Incipient encephalitis remainder frontal lobe and adjacent temporal lobe. Purulent meningitis entire right cerebral hemisphere.

**Zemann**: (1) Chr. bilateral frontal sinusitis; fistula through left eyebrow; left-sided ethmoid cell suppuration. Radical operation left side. Subperiosteal abscess cavity. Two weeks later operation right frontal sinus. Death in three days. Autopsy showed adhesion right frontal lobe to posterior surface of frontal sinus. Two deeper abscesses in frontal lobe behind superficial cortical abscess in white brain substance frontal pole and separated from cortical abscess by layer of brain tissue.

**Lubbers, K.**: (1) Chr. frontal sinusitis and fistula right eyebrow followed by osteomyelitis, frontal bone and frontal sinus empyema. Abscess right frontal lobe which author believed had existed for 18 months. Recovery.

**Rische, H.**: (1) Headache; swelling above left eye. Incised. Pus evacuated. Stupor, vomiting. External frontal sinus operation. Dura incised. Abscess found in frontal lobe. Recovery.

**Butzengeiger, C.**: (1) Infection nasopharynx and frontal sinus; pus rupturing to outside causing external abscess right side of forehead. Simultaneously inward progression of infection. Operation, right frontal sinus exposed. Frontal abscess found. Eight days later headache and vomiting. Wound re-opened twice for re-filling abscess. Hernia cerebri. Ablation. Recovery.

**White, J. A.**: (1) Chr. frontal sinusitis. Dullness of intellect. Operation disclosed frontal lobe abscess. Unconsciousness. Lost use right arm and muscles of face. Second abscess found in Rolandic fissure. Death.

**McCoy, J.**: (1) Pneumonia; swelling of eyelids one week later. Incised. Fistulous opening showed eroded bone. Double ethmoiditis perforating both orbital plates. Extension necrosis frontal and forehead regions. Osteomyelitis. Epidural abscess left upper forehead. Nausea, vomiting. Cerebral symptoms. Paralysis right side. Coma. Disintegration entire frontal lobe. Death.

**McCoy, J.** (second case): (1) Pain left eye; frontal sinus relieved considerable pus. Improvement, but almost constant headache and intermittent discharge of pus. Swelling left forehead and epidural abscess found on incision. Headache, drowsiness, nausea, vomiting. Osteomyelitis. Ethmoid labyrinth thoroughly removed. Convulsions. Wound re-opened and multiple abscesses found. Hernia cerebri. Gradual recovery.

**Hurd, L. M.**: (1) Chr. pansinusitis. Epidural abscess; frontal lobe abscess. Meningitis. Death.

**Coulter, R. J.**: (1) Trauma to left orbit (piece of wood) through upper eyelid.

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McCoy, J. (second case): (1) Pain left eye; frontal sinus relieved considerable pus. Improvement, but almost constant hea [illegible] interm [illegible] of pus. Swelling left forehead and epidura [illegible] on [illegible] ache, drowsiness, nausea, vomiting. Oste [illegible] id [illegible] bone lesions. Microscopical abscess on cortical surface of brain. Incipient osteitis orbital roof.

**Reinhard**: (1) Left-sided frontal sinus suppuration, and frontal lobe abscess. Extradural abscess. Meningitis and brain pressure after operation. Loss of consciousness. Death. Post-mortem showed behind extradural abscess, abscess on floor of second left frontal convolution. Layer of healthy brain tissue between.

**Hubbard**: (1) Ozena. Acute mastoiditis. No operation. Two months later mental dullness and headache. Mastoid tenderness: death. Post-mortem showed abscess centre frontal lobe. Dura adherent to under surface right frontal lobe.

**Linden**: (1) Empyema left frontal sinus. Brain abscess. Death. Post-mortem showed dura and pia adherent. Thick-walled abscess. No direct communication between abscess and sinus. Path of infection—sequestration of posterior wall.

**Paunz, M.**: (1) Acute left frontal sinusitis. Headache and vomiting. Dilated sluggish pupils. Venous hyperemia of eyegrounds. Operation—bulging dura. Incision of dura and brain substance; pus aspirated. Death.

**Herzfeld**: (1) Coryza. Left-sided frontal headaches. External operation. Posterior wall frontal sinus carious and removed. Epidural abscess found. Dura incised. Subdural abscess evacuated. Fistula in frontal lobe of brain. Recovery.

**Denker**: (1) Pain left temple and eye. Intranasal operation for left frontal sinusitis and ethmoiditis. One month later external operation. Headaches, somnolence, blurring left optic disc. Posterior wall frontal sinus removed. Epidural abscess exposed. Dura incised. Brain incised and abscess evacuated. Recovery.

**Trautmann**: (1) Empyema left frontal sinus. Perforation into orbit and anterior cranial fossa. Abscess left middle frontal lobe. Caries orbital roof. Death two weeks later. Post-mortem showed dura adherent to left frontal lobe and brain. Perforation of posterior wall. Circular orifice in bone communicating with frontal sinus.

**Wilson**: (1) Abscess frontal sinus and frontal lobe. Perforation external and internal wall. Death.

**Pfingst, A. O.**: (1) Ethmoidal involvement. Head pains on side of orbit. Convulsions. Sudden death. Post-mortem showed large encapsulated abscess in frontal lobe. Path of infection through blood vessels.

**Jessamon, L. W.**: (1) Tenderness over left frontal sinus and ethmoids. Nasal operation. Temperature. Thick speech. Aphasia. Paresis right arm. Complete paralysis right hand. Posterior wall frontal sinus removed. Congestion optic disc. Aphasia increased. Third operation subdural abscess. Death. Post-mortem showed dura thickened especially over ethmoid, and discoloration over ethmoid bone.

### Adjacent Frontal Lobe Abscess from Ethmoid Disease.

**Vangehuchten**: (1) Anomaly of ethmoid sinus cause of infection.

**Silex**: (1) Empyema of ethmoid cells and frontal sinus, previous to two abscesses of frontal lobes.

**Williamson**: (1) Large frontal (right) abscess after influenza, with profuse nasal discharge and frontal and occipital headache. Small amount of pus in right ethmoid cells.

**Knapp**: (1) Abscess left frontal lobe after orbital phlegmon evacuated from pus. Anterior ethmoid cells filled with offensive pus.

**Symonds**: (1) Chronic suppuration of ethmoid sinus. Death from diffuse meningitis. Post-mortem showed two abscesses under surface left frontal lobe.

Dura mater beneath frontal lobe adherent to brain. Neither abscess opened lateral ventricle. Extension to orbit.

Symonds (second case): (1) Chronic suppuration ethmoidal and frontal sinuses with abscess left frontal lobe. Influenza. Nasal discharge. Operation for suspected subdural or cerebral collection. Death. Cerebral infection from ethmoid. Extension to orbit.

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McCoy, J. (second case): (1) Pain left eye; frontal sinus relieved considerable pus. Improvement, but almost constant headache and intermittent discharge of pus. Swelling left forehead and epidural abscess found on incision. Headache, drowsiness, nausea, vomiting. Osteomyelitis. Ethmoid labyrinth thoroughly removed. Convulsions. Wound re-opened and multiple abscesses found. Section of bone removed from temporal region. Hernia cerebri. Gradual recovery.

**Hurd, L. M.**: (1) Chr. pansinusitis. Epidural abscess. Frontal lobe abscess. Meningitis. Death.

**Stucky**: (1) Traumatic ethmoiditis. Meningitis. Abscess frontal lobe. No localizing symptoms. Death. Post-mortem showed necrotic cribriform plate. Fracture through table left frontal sinus. Cerebral convolutions softened.

**Cisneros**: (1) Frontal sinusitis. Evacuation of ethmoid operation for brain abscess. Disintegration large portion right frontal lobe. Death from meningitis.

**Milligan, W.** (1) Left frontal and ethmoidal sinus disease. Supraorbital incision. Nasal drainage. Abscess left frontal lobe. Extensive basal meningitis. Death.

**Denker**: (1) Pain left temple and eye. Intranasal operation for left frontal sinusitis and ethmoiditis. One month later external operation. Headaches. Somnolence. Blurring left optic disc. Posterior wall frontal sinus removed. Epidural mass exposed. Dura incised. Brain incised and abscess evacuated. Recovery.

**Pfingst, A. O.**: (1) Ethmoidal involvement. Head pains on side of orbit. Convulsion. Sudden death. Post-mortem showed large encapsulated abscess in frontal lobe. Path of infection through blood vessels.

**Jessamon, L. W.**: (1) Tenderness over left frontal sinus and ethmoids. Nasal operation. Temperature. Thick speech. Aphasia. Paresis right arm. Complete paralysis right hand. Posterior wall frontal sinus removed. Congestion optic disc. Aphasia increased. Third operation. Subdural abscess. Death. Autopsy showed dura thickened especially over ethmoid and discoloration over ethmoid bone.

### Adjacent Frontal Lobe Abscess from Frontal Sinus.

**Cisneros**: (1) Latent sinusitis (acute) with complete obstruction of frontal canal.

**Solowcejczyk and Karbowski**: (1) Chr. latent frontal sinus suppuration with intracranial complications in the form of epidural abscess.

**Schousboe**: (1) Empyema frontal sinus and anterior ethmoid cells complicated by orbital abscess and frontal lobe abscess. Improvement after operation. Later dull and indifferent, loss of appetite, vomiting, retarded pulse. Craniotomy. Recovery.

**Moeller** (Quoted by Schousboe): (1) Left maxillary sinus empyema, orbital abscess; ethmoid cells and sphenoid cavity evacuated. During convalescence from operation sudden nausea and death. Frontal sinus empyema and frontal sinus abscess quoted as cause of death.

**Manasse**: (1) Acute frontal sinusitis followed by left frontal, left temporal and left occipital abscess.

**Eckstein**: (1) Subperiosteal abscess at lateral end of left supraorbital margin.

**Gruening**: (1) Chr. suppuration of ethmoid cells and frontal sinus after nasal pneumococcus invasion which resulted in brain abscess.

**Preysing**: (1) Left-sided frontal lobe abscess following empyema left maxillary sinus, frontal sinus and ethmoid cells from gangrenous purulent process of left nose.

**Steese**: (1) Post-mortem showed abscess in left frontal lobe and perforation in posterior wall of frontal sinus.

**Koebel**: (1) Right frontal abscess after combination of mastoid, antrum, and frontal sinus suppuration.

**Butzengeiger, O.:** (1) Infection nasopharynx and frontal sinus, pus rupturing to outside causing external abscess right side of forehead. Simultaneously inward progression of infection. Operation. Right frontal sinus exposed. Frontal abscess found. Eight days later headache, vomiting. Wound reopened for refilling of abscess. Hernia cerebri. Ablation. Recovery.

**White, J. A.:** (1) Chr. frontal sinusitis. Dullness of intellect. Operation disclosed frontal lobe abscess. Unconsciousness. Lost use right arm and muscles of face. Second abscess found in Rolandic fissure. Death.

**McCoy, J.:** (1) Pneumonia; swelling of eyelids one week later; incised. Fistulous opening showed eroded bone. Double ethmoiditis perforating both orbital plates. Extension necrosis frontal and forehead regions. Osteomyelitis. Epidural abscess left upper forehead. Nausea and vomiting. Cerebral symptoms; paralysis right side. Coma. Disintegration entire frontal lobe. Death.

**McCoy, J.** (second case): (1) Pain left eye. Frontal sinus operation; considerable pus. Improvement, but almost constant headache and intermittent discharge of pus. Swelling left forehead and epidural abscess found on incision. Headache, drowsiness, nausea, vomiting. Osteomyelitis. Ethmoid labyrinth thoroughly removed. Convulsions. Wound re-opened and multiple abscesses found. Section of bone removed from temporal region. Hernia cerebri. Gradual recovery.

**Hurd, L. M.:** (1) Chr. pansinusitis; epidural abscess; frontal lobe abscess; meningitis; death.

**Fagge, C. H.:** (1) Empyema frontal sinus. Radical operation. Headache; apathy. Subnormal temperature; abscess left frontal lobe. Death. Post-mortem showed abscess nearly to anterior horn of lateral ventricle.

**Keimer:** (1) Acute right-sided frontal sinus inflammation. Three operations. In third large abscess extending close to lateral ventricle evacuated. Death five days later.

**Lambur, M. P.:** (1) Chr. frontal sinusitis. Operation. Posterior bony wall intact. Infection of brain through venous or lymphatic channels. Abscess frontal lobe. Recovery.

**Cargill, Turner, and Thomas:** (1) Orbital cellulitis incised. Later irritability and optic neuritis. Frontal sinus opened. Posterior wall necrotic. Brain covered with granulations. Abscess frontal lobe evacuated. Recovery.

**Freudenthal, W.:** (1) Acute frontal sinusitis. Headache. Nasal discharge. Œdema right eye. Posterior wall of sinus eroded. Convulsions left side. Death several days later.

**Rawling, L. B.:** (1) Frontal headache. Œdema right upper eyelid. Coma. Paralysis left side of body and face. Vomiting. Optic neuritis. Operation. Abscess found. Recovery. Two later operations for return of symptoms. Headache. Left hemiplegia. Marked hernia. Frontal sinus contained carious bone. Recovery.

**Mayer, H.:** (1) Radical sinus operation for left-sided frontal sinus empyema. Death. Frontal abscess. Purulent meningitis. Thrombosis of sagittal and cavernous sinus. Bone intact. Infection occurred by way of blood vessels. Veins of roof of frontal sinus filled with pus and coagulated blood.

**Hammesfahr:** (1) Left-sided frontal sinus empyema and frontal lobe abscess. Symptoms of brain pressure eight weeks later. Operation. Abscess found in posterior pole of lower frontal convolution. Recovery. Infection occurred by way of venous channel.

**Roepke:** (1) Left frontal sinus and maxillary suppuration. Subperiosteal and extradural abscess. Second operation frontal lobe abscess. Death. Probable rupture into lateral ventricles.

**Roepke** (second case): (1) Bilateral frontal sinus suppuration. Osteomyelitis.

Frontal lobe abscess. Meningitis. Three operations. Abscess had ruptured into meninges and into the lateral ventricle. Death.

**Hoffmann, R.**: (1) Bilateral chr. sinus suppuration. Multiple brain abscess. Left-sided traumatism. Two operations. Recovery. Recurrence five months later. Death. Post-mortem showed infection by lymphatics.

**Goris**: (1) Chr. frontal sinusitis; fever, vomiting, convulsions. Operation. Evacuation extradural abscess. Death. Post-mortem showed frontal lobe abscess.

**Goris** (second case): (1) Chr. frontal sinusitis. Meningitis. Death three days after operation for extradural abscess. Post-mortem showed frontal lobe abscess (subdural).

**Cisneros**: (1) Frontal sinusitis. Evacuation of ethmoid. Operation for brain abscess. Disintegration large portion right frontal lobe. Death from meningitis.

**Seifert**: (1) Bilateral empyema frontal sinus. Removal nasal polyps. Death five days later. Post-mortem showed abscess right frontal lobe. Continuity infection posterior wall through lymphatics or blood vessels.

**Milligan, W.**: (1) Left frontal and ethmoidal sinus disease. Supra-orbital incisions. Nasal drainage. Abscess left frontal lobe. Extensive basal meningitis. Death.

**Milligan, W.** (second case): (1) Left frontal and maxillary sinus disease. Supra-orbital incision. Alveolar drainage. Abscess left frontal lobe. Extensive basal meningitis. Death.

**Garipuy**: (1) Acute frontal sinusitis; orbital phlegmon. Two operations. Death. Post-mortem showed abscess frontal lobe at level of meningeal and bone lesions. Microscopical abscess on cortical surface of brain. Incipient osteitis orbital roof.

**Reinhard**: (1) Left-sided frontal sinus suppuration and frontal lobe abscess. Extradural abscess. Meningitis and brain pressure after operation. Loss of consciousness. Death. Post-mortem showed behind extradural abscess one on floor of second left frontal convolution. Layer of healthy brain tissue between.

**Linden**: (1) Empyema left frontal sinus. Brain abscess. Death. Post-mortem showed dura and pia adherent. Thick-walled abscess. No direct communication between abscess and sinus. Path of infection—sequestration of posterior wall.

**Paunz, M.**: (1) Acute left frontal sinusitis. Headache and vomiting. Dilated sluggish pupils. Venous hyperemia of eyegrounds. Operation. Bulging dura; incision of dura and brain substance. Pus aspirated. Death.

**Herzfeld, J.**: (1) Coryza. Left-sided frontal headaches. External operation. Posterior wall frontal sinus carious and removed. Epidural abscess found. Dura incised. Subdural abscess evacuated. Fistula in frontal lobe of brain. Recovery.

**Denker**: (1) Pain left temple and eye. Intranasal operation for left frontal sinusitis and ethmoiditis. One month later external operation. Headache, somnolence, blurring left optic disc. Posterior wall frontal sinus removed. Epidural abscess exposed. Dura incised. Brain incised and abscess evacuated. Recovery.

**Trautmann**: (1) Empyema left frontal sinus. Perforation into orbit and anterior cranial fossa. Abscess left middle frontal lobe. Caries orbital roof. Death two weeks later. Post-mortem showed dura adherent to left frontal lobe and brain. Perforation of posterior wall. Circular orifice in bone communicating with frontal sinus.

**Wilson**: (1) Abscess frontal sinus and frontal lobe. Perforation external and internal wall. Death.

**Jessamon, L. W.:** (1) Tenderness over left frontal sinus and ethmoids. Nasal operation. Temperature; thick speech. Aphasia. Paresis right arm. Complete paralysis right hand. Posterior wall frontal sinus removed. Congestion optic disc. Aphasia increased. Third operation. Subdural abscess. Death. Post-mortem showed dura thickened, especially over ethmoid, and discoloration over ethmoid bone.

### Frontal Lobe Abscess with Orbital Complications.

**Nonne:** (1) Choked disc from neuritis optica.

**Schousboe:** (1) Empyema frontal sinus and anterior ethmoid cells complicated by orbital abscess and frontal lobe abscess.

**Moeller:** (1) Left maxillary sinus empyema and orbital abscess; ethmoid cells and sphenoid.

**Eagleton:** (1) Case XXI, D. McS., Chapter Seven, p. 111.

**Eckstein:** (1) Subperiosteal abscess at lateral end of left supra-orbital margin.

**Gruening:** (1) Chr. suppuration of ethmoid cells, pneumococcic, involving nasal, ethmoid, orbit, and brain.

**Steese:** (1) Pain in both eyes; dimness of vision and vomiting; abscess left frontal lobe.

**Steese** (second case): (1) Abscess at upper end of fissure of Rolando and another in occipital lobe.

**Lee, J. M.:** (1) Traumatic. Penetrating foreign body above eyelid (right upper) and passed through sphenoidal fissure into frontal lobe.

**von Schröder:** (1) Right frontal abscess after right retrobulbar phlegmon. Incision orbital margin; voided much thick pus.

**Kreckl:** (1) Left frontal abscess after nasal discharge of pus and polyps. Operation for swelling *above left eye* and weeping fistula. Frontal sinus filled with pus. Pain in occipital region.

**Zdekaur:** (1) Left-sided ptosis after traumatic penetrating wound of left side of head from which pus discharged.

**Redtenbacher:** (1) Abscess apex frontal lobe after abscess left upper eyelid.

**Zirm:** (1) Frontal lobe abscess coming laterally from optic nerve and showing cellular tissue of orbit congested. (Cavernous Sinus.)

**Borelius:** (1) Traumatic puncture upper eyelid. Epileptiform seizures and other cerebral symptoms. Twitching more marked left arm and leg. Incision in upper eyelid. Drainage tube; recovery.

**Williamson:** (1) Abscess right frontal lobe after influenza with profuse nasal discharge and frontal and occipital headache. Optic neuritis right eye.

**Weichselbaum:** (1) Abscess right upper eyelid. Eyeball intact. Pus in right maxillary and frontal sinus and between posterior wall of right frontal sinus and dura mater. Pachymeningitis and leptomeningitis and brain abscess.

**Renton, J. C.:** (1) Abscess right frontal lobe after incision with evacuation of pus along upper eyelid for right orbital cellulitis. Right orbital plate necrosed. No direct communication between pus from orbit and cerebral abscess.

**Knapp:** (1) Abscess left frontal lobe after orbital phlegmon which was incised and pus evacuated. Base of frontal lobe over orbital margin yellowish in color and adherent to dura mater. Some pus flakes on dura. Orbit free from pus.

**Bousquet:** (1) Acute empyema right frontal sinu- [illegible] Subperiosteal orbital abscess and necrosis frontal bone. Puncture of [illegible] bone erode[illegible] [illegible] filled with cheesy pus.

**Symonds, C. J.:** (1) Chr. suppuration ethmoi[illegible] meningitis. Post-mortem showed two abscesses [illegible] Dura mater beneath frontal lobe adherent to [illegible] lateral ventricle. Extension to orbit.

**Symonds, C. J.** (second case): (1) Chr. suppuration ethmoidal and frontal sinuses with abscess left frontal lobe. Influenza. Nasal discharge. Operation for suspected subdural or cerebral collection. Death cerebral infection from ethmoid sinus. Extension to orbit.

**Schaefer, H.**: (1) Orbital phlegmon with death from meningitis. Post-mortem showed abscess. Destruction right ethmoid labyrinth rupturing through lamina papyracea, resulting in osteitis and periostitis of orbit. Later pachymeningitis and purulent leptomeningitis. Death.

**Struycken, H. J. L.** (second case): (1) Right-sided headache with rapidly developing œdema of surroundings right eye and protrusion of eyeball. Ethmoid cells opened and most of them removed. Large abscess behind eyeball opened. Later rigidity nape of neck; vomiting. Another operation and another abscess found. Recovery with partial amnesia and persistent loss of smell on right side.

**Manasse**: (1) Acute accessory sinus suppuration which led to enormous orbital phlegmon. Frontal sinus revealed pus during after treatment. Cerebral symptoms in form of vomiting, headaches, fever. Frontal sinus diseased and closed ethmoid cell found attached to its posterior wall. Three operations and at last frontal lobe brain abscess found under this frontal ethmoid cell. Suppurative process passed from this frontal ethmoid cell to bone and then to cranial contents, through preformed vascular channels. Recovery.

**Uffenorde, W.**: (1) Ozena and left-sided frontal sinus and ethmoid suppuration leading to *orbital phlegmon* through fistula. Operations (2) revealed extradural abscess and frontal sinus abscess. Two months later frontal abscess and meningitis after rupture of frontal lobe abscess into ventricle. Death.

**Maffei**: (1) Partial loss of vision followed by vomiting and severe headache. Incipient neuroretinitis. Later pains in nape of neck and rise in temperature. Death from asphyxia. Spontaneous discharge of pus from left nostril and filling throat. No operation. No autopsy, but author believed disease to be in frontal lobe.

**Meurers**: (1) Bilateral frontal sinusitis with abscess right frontal lobe. Sudden pain left eye. Operation left frontal sinus. Recurrence severe headache and sudden death in collapse. Post-mortem showed abscess right frontal lobe with delicate membrane. Cranial sinuses free from pathological changes.

**Piffl, O.**: (1) Chr. left-sided frontal sinusitis and *exophthalmos*. Radical operation. Removal orbital roof and incision of exposed dura. Frontal lobe adherent to orbital roof and frontal abscess found at autopsy.

**Elschnig, A.**: (1) Chr. tuberculous tumor, lachrymal gland right eye. Operation, extirpation of tumor with temporary resection of lateral orbital wall. Death four weeks after. Abscess found in right frontal lobe directly above base with circumscribed adhesion of meninges.

**Elschnig, A.** (second case): (1) Phlegmon left upper eyelid incised. Paresis right side and mental apathy. Operation left frontal abscess. Removal orbital roof and dura. Recovery.

**Elschnig, A.** (third case): (1) Chr. frontal sinusitis. Incipient orbital phlegmon. Radical operation after incision at upper inner orbital margin. Abscess found in frontal brain. Death. Autopsy showed abscess reaching to area of second frontal convolution. Incipient encephalitis remainder frontal lobe and adjacent temporal lobe. Purulent meningitis entire right cerebral hemisphere.

**Zemann, W.**: (1) Chr. bilateral frontal sinusitis. Fistula through left eyebrow. Left-sided ethmoid cell suppuration. Radical operation left side. Subperiosteal abscess cavity. Two weeks later operation rt. frontal sinus. Death in three days. Autopsy showed adhesion rt. frontal lobe to posterior surface of frontal sinus. Two deeper abscesses in frontal lobe behind superficial cortical abscess in white brain-substance frontal pole, and separated from cortical abscess by layer of brain tissue.

McCoy, J. (second case): (1) Pain left eye. Frontal sinus operation; considerable pus. Improvement but almost constant headache, and intermittent discharge of pus. Swelling left forehead and epidural abscess found on incision. Headache, drowsiness, nausea and vomiting. Osteomyelitis. Ethmoid labyrinth thoroughly removed. Convulsions. Wound re-opened and multiple abscesses found. Section of bone removed from temporal region. Hernia cerebri. Gradual recovery.

Coulter, R. J.: (1) Trauma to left orbit (piece of wood) through upper eyelid. Convergent squint and diplopia. Swelling in orbit incised. Three months later apathy; loss of memory; subnormal temperature. Both eyes proptosed. Paralysis left external rectus. Choked disc. Skull trephined. Frontal lobe abscess. Month later twitching rt. arm and leg. Second abscess tapped. Recovery.

Taylor, F. L.: (1) Trauma to rt. eye. Superficial abscess opened one month later. Headache, apathy, emaciation, optic neuritis. Paresis left side of face. Stiffness back of neck. Vomiting. Incontinence of urine and feces. Operation. Rt. frontal abscess. Recovery with poor vision. Following year unconscious, convulsions, death.

Cargill, Turner, and Thomas: (1) Orbital cellulitis incised. Later irritability and optic neuritis. Frontal sinus opened. Posterior wall necrotic. Brain covered with granulations. Abscess frontal lobe evacuated. Recovery.

Reis, W.: (1) Traumatic panophthalmitis. Brain abscess. Incision of eyeball. Evacuation of pus. Headache, fever, coma; death from diffuse meningitis.

Garipuy: (1) Acute frontal sinusitis. Orbital phlegmon. Two operations. Death. Post-mortem showed abscess frontal lobe at level of meningeal and bone lesions. Microscopical abscess on cortical surface of brain. Incipient osteitis orbital roof.

Paunz, M.: (1) Acute left frontal sinusitis. Headache and vomiting. Dilated sluggish pupils. Venous hyperemia of eyegrounds. Operation. Bulging dura. Incision of dura and brain substance. Pus aspirated. Death.

Denker: (1) Pain left temple and eye. Intranasal operation for left frontal sinusitis and ethmoiditis. One month later external operation. Headache, somnolence. Blurring left optic disc. Posterior wall frontal sinus removed. Epidural abscess exposed. Dura incised. Brain incised and abscess evacuated. Recovery.

### Frontal Lobe Abscess Associated with Osteomyelitis.

Schwabach: (1) Osteitis and osteomyelitis in petrous portion of temporal bone.

Solowcejczyk, A., and Karbowski, B.: (1) Osteomyelitis of entire cranial vault.

Meurers: (1) Osteomyelitis of diploe.

Reimer: (1) Caries of frontal bone; dura adherent and greatly thickened in neighborhood of abscess left frontal lobe.

Bousquet: (1) Necrosis frontal bone and subperiosteal orbital abscess. Post-mortem showed right frontal abscess. Sinus filled with cheesy pus.

Lubbers, K.: (1) Chr. frontal sinusitis and fistula right eyebrow, followed by osteomyelitis frontal bone and frontal sinus empyema. Abscess right frontal lobe which author believes had existed for eighteen months.

McCoy, J.: (1) Pneumonia; swelling of eyelids one week later; incised. Fistulous opening showed eroded bone. Double ethmoiditis perforating both orbital plates. Extension necrosis frontal and forehead regions. Osteomyelitis. Epidural abscess left upper forehead. Nausea, vomiting. Cerebral symptoms. Paralysis right side. Coma. Disintegration entire frontal lobe. Death.

McCoy, J. (second case). (1) Pain left eye; frontal sinus operation; considerable pus. Improvement, but almost constant headache and intermittent discharge of pus. Swelling left forehead and epidural abscess found on incision. Headache, drowsiness, nausea and vomiting. Osteomyelitis. Ethmoid labyrinth thoroughly removed. Convulsions. Wound re-opened and multiple abscesses found. Section of bone removed from temporal region. Hernia cerebri. Gradual recovery.

**ROEPKE** (second case): (1) Bilateral frontal sinus suppuration. Osteomyelitis. Frontal lobe abscess. Meningitis. Three operations. Abscess had ruptured into meninges and into lateral ventricle. Death.

### METASTATIC FRONTAL LOBE ABSCESS.

**CASAMAJOR:** (1) Idiopathic double abscess frontal lobe; no history of operation. Death seven days after admission. Autopsy showed double frontal abscess primary on right side, extending through corpus callosum into left frontal lobe. Meninges showed no evidence of involvement. No diagnosis until autopsy.

**SCHWABACH:** (1) Left-sided otitis media followed by headache and vertigo. Abscess in right frontal lobe found at autopsy with "granulating congested capsule several mm. thick. Extensive purulent disintegration left petrous bone; labyrinth extensively diseased; complete rupture labyrinth capsule. Osteitis and osteomyelitis in petrous portion temporal bone. First symptom hardness of hearing left ear; five weeks later headache with disturbance of equilibrium in form of vertigo and tottering gait. Onset is not fully reported.

**LOMBARD, BLOCH, and MOULONGUET:** (1) Operation for left-sided chronic ear suppuration. Epilepsy with symptoms of intracranial hypertension suddenly five years later. Squama trephined and temporo-sphenoidal lobe punctured. Next day left-sided hemiparesis with hypesthesia and disappearance of cutaneous and tendon reflexes same side. Five days later second intervention with trephining of occipital bone and puncture of cerebellum. Death following day. Autopsy showed abscess rt. frontal lobe.

**PICQUÉ:** (1) Influenza without pulmonary complications, but with severe pain in right ear and profuse suppuration. Three months later pain returned with great severity. Violent hemicrania, progressive loss of strength, rise of temperature. Mastoid process slightly tender on pressure, and it was decided to perform exploratory trephining operation on mastoid process. Next day rise of temperature, delirium, blunted intelligence and complete left-sided hemiplegia. Mastoid process trephined; no pus. Hernia cerebri. Intracerebral abscess in front of Rolandic fissure. Autopsy showed generalized encephalitis and second smaller focus of pus behind first.

**STEESE:** (1) Metastatic from lung. Abscess at upper end of fissure of Rolando; another in occipital lobe.

**REIMER** (second case): (1) Rickets, headache, rigidity nape of neck, contracture right arm, left-sided facial paralysis, convulsions, convergent strabismus, nystagmus, fever; idiocy. Autopsy showed frontal convolutions right anterior lobe entirely flattened and almost completely transformed into abscess which had already ruptured into right lateral ventricle. Left anterior lobe contained central abscess size of walnut.

**BERENS, T. P.:** (1) Deafness rt. ear. Intermittent purulent discharge. Tender mastoid. Radical operation. Sixteen days later profuse hemorrhage from middle ear cavity. After convulsions headache, stupor, coma. Later, attack of typical Jacksonian epilepsy. Incision through dura revealed pus. Hernia of brain filling bone wound. Death some days later. Autopsy showed subdural abscess over rt. hemisphere. Large abscess also occupying most of rt. frontal lobe with spontaneous discharge into subdural space. Author believes case to be of otitic origin.

**HIRSCHBERG, O.:** (1) Metastatic brain abscess following abscess bronchial glands. Fistula from broken down glands to esophagus. Death from purulent meningitis. Autopsy showed abscess rt. frontal lobe and several smaller abscesses.

**Stucky**: (1) Acute otitis right ear. Syphilis. Metastatic abscess left frontal lobe. Very chronic as shown by firm capsule. Death from meningitis.

**Johnston**: (1) Left-sided frontal sinusitis. Operation showed healthy bone. Sudden death four weeks later. Large abscess left frontal lobe, evidently present previous to operation. Membranous capsule.

**Schorstein**: (1) Tuberculosis. Bronchiectatic cavities. Facial paralysis left side. Muscular spasm and later hemiplegia rt. side. Death three days later. Autopsy showed two abscesses frontal lobe.

**Schorstein** (second case): (1) Tuberculosis. Bronchiectatic cavities. Dullness and apathy. Death. Post-mortem showed abscess rt. frontal lobe.

**Lambur, M. P.**: (1) Chr. frontal sinusitis. Operation. Posterior bony wall intact. Infection of brain through venous or lymphatic channels. Abscess frontal lobe. Recovery.

**Wertheim**: (1) Fever; headache; discharge from nose. Tumor over right eye. Incision voided pus. Sinus wall roughened. Death. Post-mortem showed abscess right frontal lobe. Necrosis posterior wall. Circumscribed pachymeningitis.

### FRONTAL ABSCESS IN OPPOSITE LOBE (METASTATIC).

**Casamajor**: (1) Idiopathic. Primary in right frontal lobe and extending through corpus callosum to left frontal lobe.

**Schwabach**: (1) Left otitis media previous to abscess in right frontal lobe.

**Lombard, Bloch, and Moulouguet**: (1) Left chr. ear suppuration previous to right frontal abscess.

**Nonne**: (1) Left tympanic suppuration previous to right frontal abscess.

**Meurers**: (1) Left-sided frontal sinusitis previous to right-sided frontal lobe abscess.

**Heiman, Th.**: (1) Bilateral empyema, antrum of Highmore, and left-sided frontal sinusitis. Bilateral earache. Severe headache and vomiting. Sudden death. Autopsy showed extradural abscess. Right lobe and both frontal sinuses, ethmoid and maxillary sinus, filled with pus. Abscess with stalk. Diffuse meningitis.

**Stucky, A.**: (1) Acute otitis right ear. Syphilis. Metastatic abscess left frontal lobe. Very chronic as shown by firm capsule.

### FRONTAL LOBE ABSCESS OF AURAL ORIGIN (METASTATIC).

**Schwabach**: (1) Left otitis media previously to right frontal lobe abscess. Extensive disease in labyrinth.

**Lombard, Bloch, and Moulouguet**: (1) Left chronic ear suppuration previous to right frontal abscess.

**Nonne**: (1) Contraction of left tympanic membrane.

**Picqué**: (1) Intracerebral abscess after right mastoid symptoms and submeningeal collections, supervening on influenza.

**Berens**: (1) Subdural abscess in right hemisphere and second in larger portion of frontal lobe.

**Koebel**: (1) Mastoid, antrum, and frontal sinus suppuration of right side previous to abscess of right frontal lobe. Abscess referred to ear disease.

**Berens** (second case): (1) Deafness right [illegible] discharge. Tender mastoid. Radical operation. [illegible] rhage from middle ear cavity. Convulsions, h[illegible] attack of typical Jacksonian epilepsy. Inci[illegible]

Hernia of brain filling bone wound. Death some days later. Autopsy showed subdural abscess over right hemisphere. Large abscess also occupying most of right frontal lobe with spontaneous discharge into subdural space. Author believes case to be of otitic origin.

**Stucky, A.**: (1) Acute otitis right ear. Syphilis. Metastatic abscess left frontal lobe. Very chronic as shown by firm capsule. Death from meningitis.

**Hubbard**: (1) Ozena. Acute mastoiditis. No operation. Two months later mental dullness and headache. Mastoid tenderness. Death. Post-mortem showed abscess centre frontal lobe. Dura adherent to under surface right frontal lobe.

### Traumatic Frontal Lobe Abscess.

**Hoffmann**: (1) Multiple brain abscesses (4) following bilateral traumatic frontal sinus suppuration. Non-penetrating.

**Steese** (fourth case): (1) Fracture base of skull. Six months later pain over right eye. Apathy and left hemiplegia. Autopsy showed large abscess right frontal lobe.

**Steese** (sixth case): (1) Two abscesses frontal lobe. Laceration of cerebellum. Fracture of skull.

**Steese** (seventh case): (1) Bullet removed from head. Entire frontal (rt.) lobe honeycombed with abscess cavities.

**Koehler**: (1) Struck forehead against file which penetrated frontal bone. Abscess left hemisphere. Cavity communicated with opening in frontal bone and with left ventricle. Third and fourth ventricles also contained pus.

**Lee, J. M.**: (1) Fell on stub of weed which penetrated upper eyelid, and passed through sphenoidal fissure into frontal lobe of brain.

**Zdekauer**: (1) Nail penetrated left side of head resulting in right hemiplegia, left ptosis; pus wound over left temporal bone. Puncture of dura. Drainage. Recovery with paresis left hand.

**Borelius, J.**: (1) Traumatic puncture upper eyelid; epilepsy; twitching more marked left arm and leg; other cerebral symptoms. Drainage tube inserted after deep incision in upper eyelid. Recovery.

**Wyss, O.**: (1) Traumatic abscess involving entire frontal lobe. Sudden onset. Severe pain followed by twitching right arm and leg. Later paralysis right arm and leg. Fever. Enlargement of head. Coma; convulsions during which child died.

**Youl**: (1) Traumatic abscess after temporal wound with later cerebral symptoms. Vomiting, convulsions and death. Brain tissue found to be disorganized. Abscess communicated with lateral ventricle.

**Aperlo**: (1) Traumatic penetrating abscess left frontal lobe; craniectomy with recovery. Second operation which revealed solution of continuity. Drainage with gauze and glass tube. Later cerebral hernia. Recovery. Complete cicatrization.

**MacEwen, Wm.**: (1) Trauma to forehead. Unconscious. Headache, somnolence, vomiting, profuse perspiration. Patellar reflexes diminished. Pupils unequal. Paresis left internal rectus. Bilateral optic neuritis. Convulsions both arms. Operation without narcosis. Abscess frontal lobe. Gradual improvement all symptoms.

**MacEwen, Wm.** (second case): (1) Trauma of forehead. Headache; wound; roughened bone. Somnolence, vomiting, temperature, chills, convulsions. Aphasia for an hour. Paralysis rt. side improved after several hours. Diagnosis, abscess in third convolution. Operation refused. Death. Autopsy showed extradural exudate. Hyperemia of dura. Abscess frontal lobe one inch from surface.

**Kennedy, Foster**: (1) Traumatic, non-penetrating, right fronto-parietal region.

Headache and irritability. Optic neuritis left side. Slight left facial weakness. Loss of smell, right side. Gradual increasing left hemiplegia. Operation showed large abscess in frontal lobe. Recovery.

**Sachs, E.:** (1) Trauma to forehead. Dull and irritable. Paralysis right arm and leg. Lumbar puncture yielded clear fluid. Unconsciousness. Convulsions. Vessels left retina tortuous. Operation. Frontal lobe abscess found and drained. Recovery.

**Damaye, H.:** (1) Frontal abscess following fatigue and bombardment. Patient soldier under observation for asthenia and headache, with symptoms of shell-shock. Death. Autopsy showed interlobar abscess right frontal lobe. Dura adherent to pia at level of right frontal lobe.

**Coulter, R. J.:** (1) Trauma to left orbit through upper eyelid. (Piece of wood.) Convergent squint and diplopia. Swelling in orbit incised. Three months later apathy. Loss of memory, subnormal temperature. Both eyes proptosed. Paralysis left external rectus. Choked disc. Skull trephined. Frontal lobe abscess. Month later twitching right arm and leg. Second abscess tapped; recovery.

**Ferreri:** (1) Trauma left side of head. Operation showed periostitis posterior wall. Sudden death four weeks later. Post-mortem showed left frontal abscess. No symptoms during life.

**Taylor, F. L.:** (1) Trauma to right eye. Superficial abscess opened one month later. Headache, apathy, emaciation. Optic neuritis. Paresis left side of face. Stiffness back of neck. Vomiting. Incontinence, urine and feces. Operation right frontal abscess. Recovery with poor vision. Following year, unconscious. Convulsions. Death.

**Stucky:** (1) Traumatic ethmoiditis. Meningitis. Abscess frontal lobe. No localizing symptoms. Death. Post-mortem showed necrotic cribriform plate. Fracture through table left frontal sinus. Cerebral convolutions softened.

**Hoffmann, R.:** (1) Bilateral chr. sinus suppuration. Multiple brain abscesses. Left-sided traumatism. Two operations. Recovery. Recurrence five months later. Death. Post-mortem showed infection by lymphatics.

**Reis, W.:** (1) Traumatic panophthalmitis; brain abscess. Incision of eyeball. Evacuation of pus. Headache, fever, coma; death from diffuse meningitis.

### Path of Infection of Frontal Lobe Abscess.

**Casamajor:** (1) Extending through corpus callosum to left frontal lobe from right.

**Lombard, Bloch, and Moulouguet:** (1) Left otitis developing right frontal abscess. Thrombophlebitis from sinus, extending along course of a cerebral vein, or arterial or venous embolus from primary infectious focus.

**Preysing:** (1) Gangrenous suppurative process in left nose and cavities, causing a circumscribed necrosis of dura over lamina cribrosa. Pia vein passing along lower side of frontal lobe became involved and probably further on thrombotic. Entrance of infective process through this vein and the lymph tracts into frontal lobe.

**Nonne:** (1) Syphilis, alcoholism, albuminuria, and left tympanic contraction previous to rt. frontal abscess. Two months before admission to hospital headache, apathy, stupor, gradual right-sided hemiparesis. Complete paralysis rt. side after 3 months and death. Autopsy showed orchitis fibrosa left side.

**Cisneros:** (1) Through lymphatic and blood route.

**Hoffmann:** (1) Multiple traumatic (4). Three abscesses on left side of dura in brain substance—"possibly caused by way of blood vessels or lymphatics." One of four frontal lobe abscesses communicated with left ventricle.

**Meurers** (second case): (1) Second case. Left-sided frontal sinus empyema ruptured through septum and gave rise to similar empyema on rt. side. Longitudinal sinus and later parietal emissary vein were infected by way of veins and frontal diploe and closed by thrombosis.

**Zemann**: (1) By continuity. Case of meningitis. Three abscesses, one of right lobe, superficial cortical, and two communicating deep-seated abscesses.

**Vangehuchten**: (1) Anomaly of ethmoid sinus through which infection occurred. Large abscess left frontal lobe.

**Gruening**: (1) Nasal, ethmoid cells, orbit and brain.

**Preysing**: (1) Gangrenous purulent process in left nose, and cavities gave rise to circumscribed necrosis of dura and pia over lamina cribrosa; pial veins became involved in disease, and thrombosed, and infective process entered through the vein, and the concomitant lymph tracts into the frontal lobe.

**Koehler**: (1) Traumatic abscess left hemisphere. Cavity communicated with opening in frontal lobe and with left ventricle. Third and fourth ventricles also contained pus. Pus had penetrated through foramen of Monro into right ventricle.

**Lee, J. M.**: (1) Traumatic penetrating foreign body, passed through upper eyelid and sphenoidal fissure into frontal lobe of brain.

**Silex**: (1) Empyema ethmoid cells and frontal sinus, followed by two abscesses both frontal lobes. Perforation of posterior and lower wall.

**von Schroeder**: (1) Caries of orbit or ethmoid cells. Rt. frontal abscess after right retrobulbar phlegmon.

**Zirm**: (1) Frontal lobe abscess. Post-mortem showed cellular tissue of orbit congested and abscess laterally from optic nerve.

**Schindler**: (1) Left frontal abscess following inflammation of frontal sinus. Perforation of posterior upper wall. Post-mortem showed large abscess in second and third left frontal convolution.

**Borelius, J.**: (1) Traumatic puncture of upper eyelid. Epileptiform seizures and other cerebral symptoms. No paralysis, but twitching more marked left arm and leg. Incision upper eyelid and probe passed through roof of orbit and frontal lobe. Drainage. Recovery.

**Weichselbaum**: (1) Influenza. Swelling right upper eyelid; eyeball intact. Abscess rt. upper eyelid. Pus right maxillary and frontal sinus and between posterior wall of frontal sinus and dura mater. Process originated from maxillary and frontal sinus where purulent pachymeningitis and leptomeningitis developed with brain abscess.

**Renton, J. C.**: (1) Abscess rt. frontal lobe after incision with evacuation of pus along upper eyelid for right orbital cellulitis. Right orbital plate necrosed. Dura inflamed over this area. Abscess cavity lined with distinct membrane, and there was no direct communication between the pus from orbit and the cerebral abscess.

**Knapp**: (1) Abscess left frontal lobe after orbital phlegmon which was incised. Base of frontal lobe was adherent to dura mater; dura was bluish covered with pus flakes. Orbit free from pus. Communication existed between orbit and dilated frontal sinus.

**Reirner**: (1) Abscess left frontal lobe with cerebral symptoms, and bone disease. Extensive caries frontal bone.

**Reirner** (second case): (1) Abscess left anterior lobe and also abscess right lobe which was flattened, and abscess had ruptured into right lateral ventricle.

**Bousquet**: (1) Acute empyema right frontal sinus and subperiosteal orbital abscess and necrosis frontal bone. Perforation in cranial cavity (posterior and inferior wall).

**Maas, H.**: (1) Nasal polypi and nasopharyngeal polyp removed. Apathy, vertigo. Exophthalmos, delirium, death. Post-mortem showed dura at base of skull covered with pus. Abscess in frontal lobe of brain above ethmoidal region. Lamina cribrosa and upper surface of sphenoid destroyed.

**Youl**: (1) Traumatic abscess at point opposite wound in temporal region. Surrounding brain tissue much disorganized. Abscess communicated with lateral ventricle.

**Symonds, C. J.:** (1) Chr. suppuration of ethmoid sinus. Death from diffuse meningitis. Post-mortem showed two abscesses under surface left frontal lobe. Dura mater beneath frontal lobe adherent to brain. Neither abscess opened lateral ventricle. Extension to orbit.

**Symonds, C. J.** (second case): (1) Chr. suppuration ethmoidal and frontal sinuses with abscess left frontal lobe. Influenza, nasal discharge. Operation for suspected subdural or cerebral collection. Death. Cerebral infection from ethmoid. Extension to orbit.

**Schaefer, H.:** (1) Orbital phlegmon with death from meningitis. Post-mortem showed abscess. Destruction rt. ethmoid labyrinth rupturing through lamina papyracea, resulting in osteitis and periostitis of orbit.

**Struycken, H. J. L.:** (1) Severe headache; pus from right nostril. Frontal sinus opened. Cerebral symptoms. Puncture cranial cavity. Drainage. Recurrence. Incomplete recovery with persistent amnesia.

**Struycken, H. J. L.** (second case): (1) Right-sided headache with rapidly developing œdema of surroundings of right eye and protrusion of eyeball. Ethmoid cells opened—most of them removed. Large abscess behind eyeball opened; later rigidity nape of neck, vomiting. Another operation and another abscess found. Recovery with partial amnesia and persistent loss of smell right side.

**Aperlo, G.:** (1) Traumatic penetrating abscess left frontal lobe. Craniectomy with recovery. Second operation which revealed solution of continuity. Drainage with gauze and glass tube. Later cerebral hernia. Recovery; complete cicatrization.

**Manasse:** (1) Acute accessory sinus suppuration which led to enormous orbital phlegmon. Frontal sinus revealed pus during after-treatment. Cerebral symptoms in form of vomiting, headache and fever. Frontal sinus diseased and closed ethmoid cell found attached to its posterior wall. At third operation frontal lobe brain abscess found under this fronto-ethmoid cell. Suppurative process passed from frontal ethmoid cell to bone and then to cranial contents through preformed vascular channels. Recovery.

**Uffenorde, W.:** (1) Ozena and left-sided frontal sinus and ethmoid suppuration leading to orbital phlegmon, through fistula. Operations (2) revealed extradural abscess and frontal sinus abscess. Two months later frontal abscess, meningitis, after rupture of frontal abscess into ventricle. Death.

**Meurers:** (1) Bilateral frontal sinusitis with abscess rt. frontal lobe. Sudden pain left eye. Operation left frontal sinus. Recurrence severe headache and sudden death, in collapse. Post-mortem showed abscess right frontal lobe, with delicate membrane. Cranial sinuses free from pathological changes.

**Freudenthal, W.:** (1) Acute empyema frontal sinus. Diffuse suppuration frontal lobe. Two operations. Died after second. No autopsy. In second operation catheter was introduced and struck only a fluid mass, so that more than frontal lobe seemed bathed in pus.

**Freudenthal, W.** (second case): (1) Influenza frontal sinusitis. Extradural collection of pus. Abscess of frontal lobe. Two operations. Headache. Discolored bone. Abscess between it and intact dura. Recovery.

**Freudenthal, W.** (third case): (1) Acute empyema sphenoid sinus. Infection extended to other accessory sinuses with abscess in temporal lobe.

**Mertins, P.:** (1) Headache; incision for swelling of glabella. Operation subsequently showed extradural abscess. Some broken down brain tissue evacuated. Anterior wall of sinus necrosed and fistulous tract from sinus to extradural abscess.

**Grünwald, L.:** (1) Ozena, empyema both frontal sinuses. Cr[illegible]truction posterior sinus wall. Right-sided pachymeningitis. Abscess frontal lobe discovered eight days after primary operation. Recovery.

Mullin, W. V.: (1) Pain over frontal sinus and pus from nasofrontal ducts. Later pus from frontal sinuses and pain across forehead—more severe left side. Sudden death. Autopsy showed abscess left frontal lobe. Necrosis extended from surface all the way through frontal lobe and perforated into left lateral ventricle.

Berens, T. P.: (1) Pansinusitis rt. side. Radical external, frontal, ethmoidal and sphenoidal operation performed. Gradual recovery. Three years later frontal headache. Swelling of cicatrix rt. frontal region. Incision through old frontal wound revealed frontal lobe abscess. Author is convinced patient carried large collection of pus for months in frontal lobe, and that this was source of nasal trouble.

Elschnig, A.: (1) Chr. tuberculous tumor lachrymal gland right eye. Operation. Extirpation of tumor with temporary resection of lateral orbital wall. Death four weeks after. Abscess found in rt. frontal lobe directly above base with circumscribed adhesion of meninges.

Elschnig, A. (third case): (1) Chr. frontal sinusitis. Incipient orbital phlegmon. Radical operation after incision at upper inner orbital margin. Abscess found in frontal brain; death. Autopsy showed abscess reaching to area of second frontal convolution. Incipient encephalitis remainder frontal lobe and adjacent temporal lobe. Purulent meningitis entire right cerebral hemisphere.

Zemann, W.: (1) Chr. bilateral frontal sinusitis; fistula through left eyebrow; left-sided ethmoid cell suppuration. Radical operation left side. Subperiosteal abscess cavity. Two weeks later operation rt. frontal sinus. Death in three days. Autopsy showed adhesion rt. frontal lobe to posterior surface frontal sinus. Two deeper abscesses in frontal lobe behind superficial cortical abscess in white brain-substance frontal pole and separated from cortical abscess by layer of brain tissue.

Berens, T. P.: (1) Deafness rt. ear; intermittent purulent discharge. Tender mastoid. Radical operation. Sixteen days later profuse hemorrhage from middle ear cavity. Convulsions, headache, stupor, coma. Then attack typical Jacksonian epilepsy. Incision through dura revealed pus. Hernia of brain filling bone wound. Death some days later. Autopsy showed subdural abscess over right hemisphere. Large abscess also occupying most of right frontal lobe with spontaneous discharge into subdural space. Case believed to be of otitic origin.

Lubbers, K.: (1) Chr. frontal sinusitis and fistula rt. eyebrow followed by osteomyelitis frontal bone and frontal sinus empyema. Abscess rt. frontal lobe which author believed had existed for eighteen months. Recovery. Dura was reached following fistulous tract.

Hirschberg, O.: (1) Metastatic brain abscess following abscess bronchial glands. Fistula from broken down glands to esophagus. Death from purulent meningitis. Autopsy showed abscess rt. frontal lobe and several smaller abscesses.

McCoy, J.: (1) Pneumonia, swelling of eyelids one week later, incised. Fistulous openings showed eroded bone. Double ethmoiditis perforating both orbital plates. Extension necrosis frontal and forehead regions. Osteomyelitis. Epidural abscess left upper forehead. Nausea, vomiting, cerebral symptoms, paralysis rt. side, coma, disintegration entire frontal lobe. Necrosis and destruction of inner plate leading to epidural abscess, thence to frontal lobe by way of blood vessels. Abscess enlarged insidiously invading lateral ventricle. Death.

McCoy, J. (second case): (1) Pain left eye; frontal sinus relieved considerable pus. Improvement, but almost constant headache and intermittent discharge of pus. Swelling left forehead and epidural abscess found on incision. Headache, drowsiness, nausea, vomiting, osteomyelitis. Ethmoid labyrinth thoroughly removed. Convulsions. Wound re-opened and multiple abscesses found. Section of bone removed from temporal region. Hernia cerebri, marked choked disc. Gradual recovery. Necrosis and destruction of inner plate leading to epidural abscess and thence to frontal lobe by way of blood vessels.

**Fagge, C. H.:** (1) Empyema frontal sinus. Radical operation; headache, apathy. Subnormal temperature; abscess left frontal lobe. Death. Post-mortem showed abscess nearly to anterior horn of lateral ventricle.

**Keimer:** (1) Acute right frontal sinus inflammation. Three operations. In third large abscess extending close to lateral ventricle evacuated. Death five days later.

**Lambur, M. P.:** (1) Chr. frontal sinusitis. Operation. Posterior bony wall intact. Infection of brain through venous or lymphatic channels. Abscess frontal lobe. Recovery.

**Cargill, Turner, and Thomas:** (1) Orbital cellulitis incised. Later irritability and optic neuritis. Frontal sinus opened. Posterior wall necrotic. Brain covered with granulations. Abscess frontal lobe evacuated. Recovery.

**Freudenthal, W.:** (1) Acute frontal sinusitis. Headache, nasal discharge. Œdema rt. eye. Posterior wall of sinus eroded. Convulsions left side. Death several days later.

**Rawling, L. B.:** (1) Frontal headache; œdema rt. upper eyelid. Coma; paralysis left side of body and face. Vomiting; optic neuritis. Operation; abscess found; recovery. Two later operations for return of symptoms. Headache, left hemiplegia. Marked hernia. Frontal sinus contained carious bone. Recovery.

**Stucky:** (1) Traumatic ethmoiditis, meningitis, abscess frontal lobe. No localizing symptoms. Death. Post-mortem showed necrotic cribriform plate. Fracture through table left frontal sinus. Cerebral convolutions softened.

**Mayer, H.:** (1) Radical sinus operation for left-sided frontal sinus empyema. Death. Frontal abscess. Purulent meningitis. Thrombosis of sagittal and cavernous sinus. Bone intact. Infection occurred by way of blood vessels. Veins of roof of frontal sinus filled with pus and coagulated blood.

**Hammesfahr:** (1) Left-sided frontal sinus empyema and frontal lobe abscess. Symptoms of brain pressure eight weeks later. Abscess found in posterior pole of lower frontal convolution. Recovery. Infection occurred by way of venous channel.

**Roepke:** (1) Left frontal sinus and maxillary suppuration. Subperiosteal and extradural abscess. Second operation frontal lobe abscess. Death. Probable rupture into lateral ventricle.

**Roepke** (second case): (1) Bilateral frontal sinus suppuration. Osteomyelitis. Frontal lobe abscess. Meningitis. Three operations. Abscess had ruptured into meninges and into the lateral ventricle. Death.

**Hoffmann, R.:** (1) Bilateral chronic sinus suppuration. Multiple brain abscess. Left-sided traumatism. Two operations. Recovery. Recurrence five months later. Death. Post-mortem showed infection by lymphatics.

**Seifert:** (1) Bilateral empyema frontal sinus. Removal nasal polyps. Death five days later. Post-mortem showed abscess rt. frontal lobe. Continuity infection posterior wall through lymphatics or blood vessels.

**Garipuy:** (1) Acute frontal sinusitis. Orbital phlegmon. Two operations. Death. Post-mortem showed abscess frontal lobe at level of meningeal and bone lesions. Microscopical abscess on cortical surface of brain. Incipient osteitis orbital roof.

**Reinhard:** (1) Left-sided frontal sinus suppuration and frontal lobe abscess. Extradural abscess, meningitis, and brain p[illegible] after ope[illegible] of consciousness. Death. Post-mortem show[illegible]xtrad on floor of second left frontal convoluti[illegible]ea between.

**Linden**: (1) Empyema left frontal sinus. Brain abscess. Death. Post-mortem showed dura and pia adherent. Thick-walled abscess. No direct communication between abscess and sinus. Path of infection sequestration of posterior wall.

**Herzfeld, J.**: (1) Coryza. Left-sided frontal headache. External operation. Posterior wall frontal sinus carious and removed. Epidural abscess found. Dura incised. Subdural abscess evacuated. Fistula in frontal lobe of brain. Recovery.

**Wertheim**: (1) Fever, headache, discharge from nose. Tumor over rt. eye. Incision voided pus. Sinus wall roughened. Death. Post-mortem showed abscess rt. frontal lobe, necrosis posterior wall. Circumscribed pachymeningitis.

**Denker**: (1) Pain left temple and eye. Intranasal operation for left frontal sinusitis and ethmoiditis. One month later external operation. Headaches, somnolence, blurring left optic disc. Posterior wall frontal sinus removed. Epidural abscess exposed. Dura incised. Brain incised and abscess evacuated. Recovery.

**Trautmann**: (1) Empyema left frontal sinus. Perforation into orbit and anterior cranial fossa. Abscess left middle frontal lobe. Caries orbital roof. Death two weeks later. Post-mortem showed dura adherent to left frontal lobe and brain. Perforation of posterior wall. Circular orifice in bone communicating with frontal sinus.

**Wilson**: (1) Abscess frontal sinus and frontal lobe. Perforation external and internal wall. Death.

**Pfingst**: (1) Ethmoidal involvement, head pains on side of orbit; convulsion, sudden death. Post-mortem showed large encapsulated abscess in frontal lobe. Path of infection through blood vessels.

### Frontal Lobe Abscess Associated with Complications.

**Lombard, Bloch, and Moulouguet**: (1) Operation right frontal lobe which yielded no pus. Day after left-sided hemiparesis with hypesthesia and disappearance of cutaneous and tendon reflexes same side. Second operation no abscess found, but autopsy showed right frontal abscess.

**Nonne**: (1) Right-sided hemiparesis of character of organic paralysis. Patellar reflex and Achilles jerk absent on both sides. Choked disc from neuritis optica. Disturbances in co-ordination, in standing and walking; ataxia of trunk muscles; absence sense of smell. Complete paralysis of right side before death.

**Solowcyczyk, A., and Karbowski, B.**: (1) Intracranial.

**Legaard**: (4) Intracranial.

**Schousboe**: (1) Empyema of frontal sinus and anterior ethmoidal cells. Patient became dull and indifferent. Loss of appetite; vomiting; retarded pulse.

**Moeller**: (1) Left maxillary sinus empyema and orbital abscess. Nauseated after operation. Ethmoid cells and sphenoid.

**Manasse**: (1) Frontal sinusitis. Intracranial complications short time after acute influenza; sinusitis. Left frontal abscess, then left temporal; then intrameningeal abscess in left occipital lobe.

**Eckstein**: (1) Somnolence. Suppurative thrombosis of longitudinal sinus with extradural abscess.

**Picqué**: (1) Severe pains right ear. Mastoid tender on pressure. Intelligence blunted. Complete left-sided hemiplegia. Cerebral hernia. Generalized encephalitis at autopsy. Smaller focus in addition to first also found.

**Steese**: (1) Pain both eyes; dimness of vision. Mental dullness; delirium, stupor. Difficulty in swallowing and breathing. Frontal sinus disease shown by post-mortem. Vomiting; urinary incontinence; constipation. Death.

**Steese** (second case): (1) Phthisis several months. Paralytic stroke. Left hemiplegia. Occasional chronic spasm left side with spasmodic conjugate deviation. Ankle clonus.

**Steese** (third case): (1) Fracture of skull; vomiting; shallow respiration. Abscess right frontal lobe.

**Steese** (fourth case): (1) Abscess in left frontal lobe. Entire lobe softened. Headache; malaise; and chills.

**Steese** (fifth case): (1) Fracture of skull. Spasms left side of face and extending to leg and arm. Abscess frontal lobe. Laceration of cerebellum. Pneumonia.

**Steese** (sixth case): (1) Bullet removed from head. Headache; stupor; twitching right hand and leg. Slight paralysis left side. Difficulty in swallowing. Right pupil dilated and left contracted. Rigor.

**Koehler**: (1) Traumatic (penetrating) abscess left hemisphere. Vomiting. Coma. Swelling. Tremor left half of body.

**Koebel**: (1) Post-mortem showed right cerebral hemisphere distinctly enlarged, as compared to left. Cavity at apex and base of medulla right frontal lobe.

**Lee, J. M.**: (1) Traumatic (penetrating)—foreign body. Convulsions. Spasms. Bulging dura.

**Rafin**: (1) Right frontal sinusitis and brain abscess. Congestion of brain.

**Tratel**: (1) Abscess frontal lobe after purulent discharge left nose and pain in forehead and cheek. Removal of polyps left nose. Hemiparesis right side. Brain hernia. Convulsions. Removal of ethmoids. Death.

**von Schroeder**: (1) Rt. frontal abscess after right retrobulbar phlegmon. Serous meningitis right hemisphere. Necrosis orbital roof. Dura under abscess inflamed.

**Krecki**: (1) Left frontal abscess after discharge of pus and polyps of nose. Operation for swelling above left eye and frontal sinus found filled with pus. Chills, fever, left-sided headache. Pain in occipital region. Apathy. Thickening of dura mater. Soft meninges at base covered with pus.

**Zdekauer**: (1) Puncture of dura after penetrating traumatic abscess left side of head. Dura found to be fluctuating. Recovery with paresis left hand.

**Redtenbacher**: (1) Abscess apex frontal lobe after abscess left upper eyelid succeeding influenza. Chronic spasms. Headache; weakness right half of body. Apathy; rigidity of neck. Pus in frontal sinus and necrosis of its posterior wall. Dura adherent to this wall.

**Borelius, J.**: (1) Traumatic puncture upper eyelid resulting in epileptiform and cerebral symptoms. Twitching more marked left arm and leg. Deep incision in eyelid. Drainage tube. Recovery.

**Williamson**: (1) Abscess frontal lobe after influenza. Nasal discharge. Frontal and occipital headache. Mentality dull which became progressively more marked. Optic neuritis right eye.

**Weichselbaum**: (1) Influenza; swelling rt. upper eyelid; eyeball intact. Headache, vomiting, delirium. Abscess right upper eyelid. Pus right maxillary and frontal sinus and dura mater. Pachymeningitis and leptomeningitis.

**Sillar**: (1) Empyema frontal sinus. Long standing sinusitis. Abscess in frontal sinus operated. Abscess frontal lobe.

**Renton, J. C.**: (1) Right frontal abscess after incision with evacuation of pus over inner side of upper eyelid for right orbital cellulitis. Later severe headache, vomiting. Spasms left side. Post-mortem convolutions over right lobe slightly flattened. Right orbital plate necrosed and dura inflamed over this area. No direct communication between pus from orbit and cerebral abscess.

**Knapp**: (1) Abscess left frontal lobe after orbital phlegmon, which was incised and pus found. Headache and vomiting. Orbit found free from pus and communication existed between orbit and dilated frontal sinus.

**Reiner**: (1) Abscess left frontal lobe with rickets and cerebral symptoms.

Headache and anesthesia left face. Diminished hearing left side and paralysis left sixth nerve.

REINER (second case): (1) Abscess both anterior lobes. Right was flattened and abscess was ruptured into right lateral ventricle. Left contained a central abscess. Headache, rigidity of neck. Contracture right arm. Left-sided facial paralysis. Convulsions. Nystagmus. Dilated pupils. Idiocy.

BOUSQUET: (1) Acute empyema right frontal sinus. Subperiosteal orbital abscess and necrosis frontal bone. Puncture of orbit. Bone eroded. Two weeks later contractures, severe œdema, loss of consciousness. Trephining frontal lobe before death.

JACUBASCH: (1) Abscess right frontal lobe. Sore in nose; dull headache; vertigo, vomiting. Speech slow and difficult. Apathy. Pain back of head. Left-sided hemiparesis.

WYSS, O.: (1) Traumatic abscess of entire frontal lobe. Sudden onset of severe pain and twitching right arm and leg. Later paralysis right arm and leg. Fever. Enlargement of head. Coma; dilated pupils. Convulsions during one of which child died.

MAAS, H.: (1) Removal of polypi and nasopharyngeal polyp followed by apathy, vertigo, exophthalmos, delirium, death. Post-mortem showed abscess in frontal lobe of brain above ethmoidal region.

YOUL: (1) Traumatic abscess in region opposite temporal wound. Cerebral symptoms two weeks after injury. Vomiting. Convulsions; death. Post-mortem showed surrounding brain tissue disorganized.

SYMONDS, C. J.: (1) Chr. suppuration ethmoid sinus. Death from diffuse meningitis. Post-mortem showed two abscesses under surface left frontal lobe. Dura mater beneath frontal lobe adherent to brain. Neither abscesses opened lateral ventricle. Extension to orbit.

SYMONDS, C. J. (second case): (1) Chr. suppuration ethmoidal and frontal sinuses with abscess left frontal lobe. Influenza. Nasal discharge. Operation for suspected subdural or cerebral collection. Death. Cerebral infection from ethmoid sinus. Extension to orbit.

SCHAEFER, H.: (1) Orbital phlegmon with death from meningitis. Post-mortem showed abscess. Destruction right ethmoid labyrinth rupturing through lamina papyracea, resulting in osteitis and periostitis of orbit. Later pachymeningitis and purulent leptomeningitis. Death.

STRUYCKEN, H. J. L.: (1) Severe headache. Pus from right nostril frontal sinus opened. Cerebral symptoms. Puncture cranial cavity. Drainage. Recurrence brain symptoms. Later a second abscess opened. Incomplete recovery with persistent amnesia.

STRUYCKEN, H. J. L. (second case): (1) Right-sided headache with rapidly developing œdema of surroundings of right eye and protrusion of eyeball. Ethmoid cells opened and most of them removed. Large abscess behind eyeball opened. Later rigidity nape of neck. Vomiting. Another operation and another abscess found. Recovery with partial amnesia and persistent loss of smell right side.

APERLO, G.: (1) Traumatic penetrating abscess left frontal lobe. Craniectomy with recovery. Second operation which revealed solution of continuity. Drainage with gauze and glass tube. Later cerebral hernia. Recovery. Complete cicatrization.

MANASSE: (1) Acute accessory sinus suppuration which led to enormous orbital phlegmon. Frontal sinus revealed pus during after-treatment. Cerebral symptoms; vomiting, headache, fever. Frontal sinus diseased and closed ethmoid cell found attached to its posterior wall. Three operations and at last frontal lobe brain abscess found under the fronto-ethmoid cell. Suppurative process passed from ethmoid cell to bone and then to cranial contents through preformed vascular channels. Recovery.

**Uffenorde, W.:** (1) Ozena and left-sided frontal sinus and ethmoid suppuration leading to orbital phlegmon through fistula. Two operations revealed extradural abscess and frontal sinus abscess. Two months later frontal abscess, meningitis, after rupture frontal abscess into ventricle. Death.

**Wiener, A.:** (1) Persistent headache. Empyema frontal sinus, right side, with two operations. Later frontal lobe abscess. Operated. Recovery.

**Hosch, P. H.:** (1) Bilateral maxillary sinusitis and right-sided ethmoidal and frontal sinusitis. Extradural perisinus abscess. Frontal lobe abscess found at base of right frontal brain only after death, because of unusual location. Purulent meningitis.

**Maffei:** (1) Partial loss of vision followed by vomiting and severe headache. Incipient neuroretinitis. Later pain in nape of neck and rise of temperature. Death from asphyxia. Spontaneous discharge from left nostril and filling throat. No operation; no autopsy, but author believed disease to be in frontal lobe.

**Meurers:** (1) Bilateral frontal sinusitis with abscess right frontal lobe. Sudden pain left eye. Operation left frontal sinus. Recurrence severe headache and sudden death in collapse. Post-mortem showed abscess right frontal lobe. Abscess with delicate membrane. Cranial sinuses free from pathological changes.

**Freudenthal, W.:** (1) Acute empyema frontal sinus. Diffuse suppuration frontal lobe. Two operations. Died after second. No autopsy. In second operation catheter was introduced and struck only a fluid mass so that more than the frontal lobe semed to be bathed in pus.

**Freudenthal, W. (second case):** (1) Influenza, frontal sinusitis. Extradural collection of pus. Abscess frontal lobe. Two operations. Headaches. Discolored bone. Abscess between it and intact dura. Recovery.

**Freudenthal, W. (third case):** (1) Acute empyema sphenoid sinus. Infection extended to other accessory sinuses with abscess in temporal lobe.

**Mertins, P. S.:** (1) Headaches. Incision for swelling of glabella. Necrosed bone. Operation subsequently showed extradural abscess. Some broken down brain tissue. Fistula. Death two weeks after operation from pulmonary complications.

**Heiman, Th.:** (1) Bilateral empyema antrum of Highmore and left-sided frontal sinusitis. Bilateral earache. Severe headache and vomiting. Sudden death. Autopsy showed extradural abscess, right lobe and both frontal sinuses, ethmoid and maxillary sinus filled with pus. Abscess with stalk. Diffuse meningitis.

**Mullin, W. V.:** (1) Pain over frontal sinuses and pus from nasofrontal ducts. Later pus from frontal sinuses and pain across forehead, more severe left side. Sudden death. Autopsy showed abscess left frontal lobe. Necrosis extended from surface all way through frontal lobe, perforating left lateral ventricle.

**Berens, T. P.:** (1) Pansinusitis rt. side. Radical external frontal ethmoidal and sphenoidal operation performed. Gradual recovery. Three years later frontal headache. Swelling of cicatrix right frontal region. Incision through old frontal wound revealed frontal lobe abscess. Author is convinced patient carried large collection of pus for months in frontal lobe and that this was source of nasal trouble.

**Piffl, O.:** (1) Chr. left-sided frontal sinusitis and exophthalmos. Radical operation. Removal orbital roof. Incision exposed dura. Frontal lobe adherent to orbital roof and frontal abscess [illegible] t autopsy.

**Elschnig, A.:** (1) Chr. tuberculous [illegible] al gla [illegible] tion, extirpation of tumor with tempor [illegible] ter [illegible] th four weeks after. Abscess found i [illegible] d circumscribed adhesions of menin [illegible]

**Elschnig, A.** (second case): (1) Phlegmon left upper eyelid incised. Paresis rt. side and mental apathy. Operation left frontal abscess. Removal orbital roof and dura. Recovery.

**Elschnig, A.** (third case): (1) Chr. frontal sinusitis. Incipient orbital phlegmon. Radical operation after incision at upper inner orbital margin. Abscess found in frontal brain. Death. Autopsy showed abscess reaching to area of second frontal convolution. Incipient encephalitis remainder frontal lobe and adjacent temporal lobe. Purulent meningitis entire rt. cerebral hemisphere.

**Grünwald, L.:** (1) Ozena; empyema both frontal sinuses. Carious destruction posterior sinus wall. Right-sided pachymeningitis. Abscess frontal lobe discovered eight days after primary operation. Recovery.

**Zemann, W.:** (1) Chr. bilateral frontal sinusitis; fistula through left eyebrow. Left-sided ethmoid cell suppuration. Radical operation left side. Subperiosteal abscess cavity. Two weeks later operation rt. frontal sinus. Death in three days. Autopsy showed adhesions rt. frontal lobe to posterior surface of frontal sinus. Two deeper abscesses in frontal lobe behind superficial cortical abscess in white brain-substance frontal pole and separated from cortical abscess by layer of brain tissue.

**Berens, T. P.:** (1) Deafness rt. ear. Intermittent purulent discharge. Tender mastoid. Radical operation. Sixteen days later profuse hemorrhage from middle ear cavity. Convulsions, headache, stupor, coma. Then attack typical Jacksonian epilepsy. Incision through dura revealed pus. Hernia of brain filling bone wound. Death some days later. Autopsy showed subdural abscess over right hemisphere. Large abscess also occupying most of rt. frontal lobe, with spontaneous discharge into subdural space. Author believes case of otitic origin.

**MacEwen, Wm.:** (1) Trauma to forehead; unconscious; headaches; somnolence, vomiting, profuse perspiration. Patellar reflexes diminished. Pupils unequal. Paresis left internal rectus. Bilateral optic neuritis. Convulsions both arms. Operation without narcosis. Abscess frontal lobe. Gradual improvement all symptoms.

**MacEwen, Wm.** (second case): (1) Trauma of forehead; headache; wound; roughened bone. Somnolence. Vomiting, temperature, chills, convulsions. Aphasia for an hour. Paralysis rt. side improved after several hours. Diagnosis—abscess in third convolution. Operation refused. Death. Autopsy showed extradural exudate; hyperemia of dura. Abscess frontal lobe one inch from surface.

**Lubbers, K.:** (1) Chr. frontal sinusitis and fistula rt. eyebrow followed by osteomyelitis frontal bone and frontal sinus empyema. Abscess rt. frontal lobe which author believed had existed for 18 months. Recovery.

**Hirschberg, O.:** (1) Metastatic brain abscess following abscess bronchial glands. Fistula from broken down glands to esophagus. Death from purulent meningitis. Autopsy showed abscess rt. frontal lobe and several smaller abscesses.

**Kennedy, Foster:** (1) Traumatic non-penetrating right fronto-parietal region. Headache and irritability. Optic neuritis left side. Slight left facial weakness. Loss of smell right side. Gradually increasing left hemiplegia. Operation showed large abscess in frontal lobe; recovery.

**Sachs, E.:** (1) Trauma to forehead. Dull and irritable. Paralysis rt. arm and leg. Lumbar puncture yielded clear fluid. Unconsciousness; convulsions. Vessels left retina tortuous. Operation. Frontal lobe abscess found and drained. Recovery.

**Rische, H.:** (1) Headache. Swelling above left eye. Incised. Pus evacuated. Stupor, vomiting, external frontal sinus operation. Dura incised. Abscess found in frontal lobe; recovery.

**Butzengeiger, O.:** (1) Infection nasopharynx and frontal sinus; pus rupturing to outside causing external abscess right side of forehead. Simultaneously inward progression of infection. Operation. Right frontal sinus exposed. Frontal abscess found. Eight days later headache and vomiting; wound re-opened twice for refilling of abscess. Hernia cerebri. Ablation. Recovery.

White, J. A.: (1) Chr. frontal sinusitis. Dullness of intellect. Operation disclosed frontal lobe abscess. Unconsciousness. Loss use rt. arm and muscles of face. Second abscess found in Rolandic fissure. Death.

Damaye, H.: (1) Frontal abscess following fatigue and bombardment. Patient soldier under observation for asthenia and headache with symptoms of shell shock. Death. Autopsy showed interlobar abscess rt. frontal lobe. Dura adherent to pia at level of rt. frontal lobe.

Stucky, A.: (1) Acute otitis rt. ear. Syphilis. Metastatic abscess left frontal lobe. Very chronic as shown by firm capsule. Death from meningitis.

McCoy, J.: (1) Pneumonia. Swelling of eyelids one week later, incised. Fistulous openings showed eroded bone. Ethmoiditis perforating orbital plate, similar condition rt. and left. Extensive necrosis frontal and forehead regions. Osteomyelitis. Epidural abscess left upper forehead. Nausea and vomiting. Cerebral symptoms; paralysis rt. side. Coma. Encephaloscope showed entire frontal lobe had disintegrated. Death.

McCoy, J. (second case): (1) Pain left eye. Frontal sinus relieved considerable pus. Improvement, but almost constant headache and intermittent discharge of pus. Swelling left forehead and epidural abscess found on incision. Headache. Drowsiness. Nausea and vomiting. Ethmoid labyrinth thoroughly removed. Convulsions. Wound re-opened and multiple abscesses found. Section of bone removed from temporal region. Hernia cerebri. Marked choked disc. Gradual recovery.

Hurd, L. M.: (1) Chr. pansinusitis; epidural abscess; frontal lobe abscess. Meningitis. Death.

Coulter, R. J.: (1) Trauma to left orbit (piece of wood) through upper eyelid. Convergent squint and diplopia. Swelling in orbit incised. Three months later apathy. Loss of memory. Subnormal temperature. Both eyes proptosed. Paralysis left external rectus. Choked disc. Skull trephined. Frontal lobe abscess. Month later twitching right arm and leg. Second abscess tapped. Recovery.

Fagge, C. H.: (1) Empyema frontal sinus. Radical operation. Headache. Apathy. Subnormal temperature. Abscess left frontal lobe. Death. Post-mortem showed abscess nearly to anterior horn of lateral ventricle.

Ferreri: (1) Trauma left side of head. Operation showed periostitis posterior wall. Sudden death four weeks later. Post-mortem showed left frontal abscess. No symptoms during life.

Johnston: (1) Left-sided frontal sinusitis. Operation showed healthy bone. Sudden death four weeks later. Large abscess left frontal lobe, evidently present previous to operation. Membranous capsule.

Keimer: (1) Acute rt. frontal sinus inflammation. Three operations. In third large abscess extending close to lateral ventricle evacuated. Death five days later.

Schorstein: (1) Tuberculosis. Bronchiectatic cavities. Facial paralysis left side. Muscular spasm and later hemiplegia rt. side. Death three days later. Autopsy showed two abscesses frontal lobe.

Schorstein (second case): (1) Tuberculosis; bronch[illegible] cavities. Dullness and apathy. Death. Post-mortem showed absces[illegible] lob[illegible]

Lambur, M. P.: (1) Chr. frontal sinusitis. Op[illegible] wall intact. Infection of brain through venous or [illegible] frontal lobe. Recovery.

Taylor, F. L.: (1) Trauma to rt. eye. Superfic[illegible] later. Headache, apathy, emaciation. Optic neur[illegible] Stiffness back of neck. Vomiting; incontinence [illegible]

Rt. frontal abscess. Recovery with poor vision. Following year unconscious, convulsions. Death.

Cargill, Turner, and Thomas: (1) Orbital cellulitis incised. Later irritability and optic neuritis. Frontal sinus opened. Posterior wall necrotic. Brain covered with granulations. Abscess frontal lobe evacuated. Recovery.

Freudenthal, W.: (1) Acute frontal sinusitis. Headache; nasal discharge. Œdema right eye. Posterior wall of sinus eroded. Convulsions left side. Death several days later.

Rawling, L. B.: (1) Frontal headache. Œdema rt. upper eyelid. Coma. Paralysis left side of body and face. Vomiting. Optic neuritis. Operation. Abscess found. Recovery. Two later operations for return of symptoms. Headache, left hemiplegia; marked hernia. Frontal sinus contained carious bone. Recovery.

Stucky: (1) Traumatic ethmoiditis; meningitis. Abscess frontal lobe. No localizing symptoms. Death. Post-mortem showed necrotic cribriform plate. Fracture through table left frontal sinus. Cerebral convolutions softened.

Mayer, H.: (1) Radical sinus operation for left-sided frontal sinus empyema. Death. Frontal abscess. Purulent meningitis. Thrombosis of sagittal and cavernous sinus. Bone intact. Infection occurred by way of blood vessels. Veins of roof of frontal sinus filled with pus and coagulated blood.

Hammesfahr: (1) Left-sided frontal sinus empyema and frontal lobe abscess. Symptoms of brain pressure eight weeks later. Operation. Abscess found in posterior pole of lower frontal convolution. Recovery. Infection occurred by way of venous channel.

Roepke: (1) Left frontal sinus and maxillary suppuration. Sub-periosteal and extradural abscess. Second operation. Frontal lobe abscess. Death. Probable rupture into lateral ventricles.

Roepke (second case): (1) Bilateral frontal sinus suppuration. Osteomyelitis. Frontal lobe abscess. Meningitis. Three operations. Abscess had ruptured into meninges and into the lateral ventricle. Death.

Hoffmann, R.: (1) Bilateral chr. sinus suppuration. Multiple brain abscess. Left-sided traumatism. Two operations. Recovery. Recurrence five months later. Death. Post-mortem showed infection by lymphatics.

Gobis: (1) Chr. frontal sinusitis. Fever, vomiting, convulsions. Operation—evacuation extradural abscess. Death. Post-mortem showed frontal lobe abscess.

Gobis (second case): (1) Frontal sinusitis; meningitis. Death three days after operation for extradural abscess. Post-mortem showed frontal lobe abscess.

Cisneros: (1) Frontal sinusitis. Evacuation of ethmoid. Operation for brain abscess. Disintegration large portion rt. frontal lobe. Death from meningitis.

Siefert: (1) Bilateral empyema frontal sinus. Removal nasal polyps. Death five days later. Post-mortem showed abscess rt. frontal lobe. Continuity infection posterior wall through lymphatics or blood vessels.

Reis, W.: (1) Traumatic panophthalmitis. Brain abscess. Incision eyeball. Evacuation of pus. Headache, fever, coma, death from diffuse meningitis.

Milligan, W.: (1) Left frontal and ethmoidal sinus disease. Supraorbital incisions. Nasal drainage. Abscess left frontal lobe. Extensive basal meningitis. Death.

Milligan, W. (second case): (1) Left frontal and maxillary sinus disease. Supraorbital incision. Alveolar drainage. Abscess left frontal lobe. Extensive basal meningitis. Death.

Garipuy: (1) Acute frontal sinusitis. Orbital phlegmon. Two operations. Death. Post-mortem showed abscess frontal lobe at level of meningeal and bone lesions. Microscopical abscess on cortical surface of brain. Incipient osteitis orbital roof.

Reinhard: (1) Left-sided frontal sinus suppuration and frontal lobe abscess.

Extradural abscess. Meningitis and brain pressure after operation. Loss of consciousness. Death. Post-mortem showed behind extradural abscess one on floor of second left frontal convolution. Layer of healthy brain tissue between.

**Hubbard**: (1) Ozena; acute mastoiditis. No operation. Two months later mental dullness and headache. Mastoid tenderness. Death. Post-mortem showed abscess centre frontal lobe. Dura adherent to under surface right frontal lobe.

**Linden**: (1) Empyema left frontal sinus. Brain abscess. Death. Post-mortem showed dura and pia adherent. Thick-walled abscess. No direct communication between abscess and sinus. Path of infection—sequestration of posterior wall.

**Paunz**: (1) Acute left frontal sinusitis. Headache and vomiting. Dilated sluggish pupils. Venous hyperemia of eyegrounds. Operation—bulging dura. Incision of dura and brain substance. Pus aspirated. Death.

**Herzfeld, J.**: (1) Coryza. Left-sided frontal headache. External operation. Posterior wall frontal sinus carious and removed. Epidural abscess found. Dura incised. Subdural abscess evacuated. Fistula in frontal lobe of brain. Recovery.

**Wertheim**: (1) Fever, headache, discharge from nose. Tumor over rt. eye. Incision voided pus. Sinus wall roughened. Death. Post-mortem showed abscess rt. frontal lobe. Necrosis posterior wall. Circumscribed pachymeningitis.

**Denker**: (1) Pain left temple and eye. Intranasal operation for left frontal sinusitis and ethmoiditis. One month later external operation. Headaches, somnolence, blurring left optic disc. Posterior wall frontal sinus removed. Epidural abscess exposed. Dura incised. Brain incised and abscess evacuated. Recovery.

**Trautmann**: (1) Empyema left frontal sinus. Perforation into orbit and anterior cranial fossa. Abscess left middle frontal lobe. Caries orbital roof. Death two weeks later. Post-mortem showed dura adherent to left frontal lobe and brain. Perforation of posterior wall. Circular orifice in bone communicating with frontal sinus.

**Wilson**: (1) Abscess frontal sinus and frontal lobe. Perforation external and internal wall. Death.

**Pfingst, A. O.**: (1) Ethmoidal involvement. Head pains on side of orbit. Convulsions. Sudden death. Post-mortem showed large encapsulated abscess in frontal lobe. Path of infection through blood vessels.

**Jessamon, L. W.**: (1) Tenderness over left frontal sinus and ethmoids. Nasal operation. Temperature. Thick speech. Aphasia. Paresis rt. arm. Complete paralysis rt. hand. Posterior wall frontal sinus removed. Congestion optic disc. Aphasia increased. Third operation, subdural abscess. Death. Post-mortem showed dura thickened especially over ethmoid and discoloration over ethmoid bone.

### Frontal Lobe Abscess Associated with Epidural Abscess.

**Hurd, L. M.**: (1) Chronic pansinusitis; epidural abscess; frontal lobe abscess; meningitis; death.

**Herzfeld, J.**: (1) Coryza. Left-sided frontal headache. External operation. Posterior wall frontal sinus carious and removed. Epidural abscess found. Dura incised. Subdural abscess evacuated. Fistula in frontal lobe of brain. Recovery.

**Denker**: (1) Pain left temple and eye. Intranasal operation for left frontal sinusitis and ethmoiditis. One month later external operation. Headaches. Somnolence; blurring left optic disc. Posterior wall frontal [illegible]d; epidural abscess exposed. Dura incised. B[illegible] and a[illegible] Recovery.

### FRONTAL LOBE ABSCESS ASSOCIATED WITH EXTRADURAL ABSCESS.

**REINHARD**: (1) Extradural abscesses discovered at time of operation associated with one on floor of second left frontal convolution.

**EAGLETON**: (1) Case XXI—D. McS., Chapter Seven, page 111.

**ECKSTEIN**: (1) Subperiosteal abscess at lateral end of left supraorbital margin with suppurative thrombosis of longitudinal sinus, and extradural abscess over upper half left frontal lobe.

**UFFENORDE**: (1) Ozena and left-sided frontal sinus and ethmoid suppuration, leading to orbital phlegmon, through fistula. Operations (2) revealed extradural abscess and frontal sinus abscess. Two months later frontal abscess, meningitis, after rupture of frontal lobe abscess into ventricle. Death.

**HOSCH, P. H.**: (1) Bilateral maxillary sinusitis and right-sided frontal and ethmoidal sinusitis. Extradural perisinus abscess, frontal lobe abscess found at base of right frontal brain after death. Purulent meningitis.

**FREUDENTHAL, W.** (second case): (1) Influenza; frontal sinusitis. Extradural collection of pus. Abscess frontal lobe. Two operations. Headaches. Discolored bone. Abscess between it and intact dura. Recovery.

**MERTINS, P.**: (1) Headaches. Incision for swelling of glabella. Necrosed bone. Subsequent operation showed extradural abscess. Some broken down brain tissue evacuated. Fistula. Death two weeks after operation from pulmonary complications.

**HEIMAN, TH.**: (1) Bilateral empyema antrum of Highmore and left-sided frontal sinusitis. Bilateral earache. Severe headache and vomiting. Sudden death. Autopsy showed extradural abscess. Right lobe and both frontal sinuses, ethmoid and maxillary sinus, filled with pus. Abscess with stalk. Diffuse meningitis.

**MACEWEN, WM.** (second case): (1) Trauma of forehead; headache; wound, roughened bone. Somnolence, vomiting, temperature, chills. Convulsions. Paralysis right side. Aphasia for an hour. Paralysis improved after several hours. Diagnosis—abscess in third convolution. Operation refused. Death. Autopsy showed extradural exudate. Hyperemia of dura. Abscess frontal lobe one inch from surface.

**ROEPKE**: (1) Left frontal sinus and maxillary suppuration. Subperiosteal and extradural abscess. Second operation frontal lobe abscess. Death. Probable rupture into lateral ventricle.

**GORIS**: (1) Chr. frontal sinusitis. Fever, vomiting, convulsions. Operation. Evacuation extradural abscess. Death. Post-mortem showed frontal lobe abscess.

**GORIS** (second case): (1) Frontal sinusitis; meningitis. Death three days after operation for extradural abscess. Post mortem showed frontal lobe abscess.

**REINHARD**: (1) Left-sided frontal sinus suppuration and frontal lobe abscess. Extradural abscess. Meningitis and brain pressure after operation. Loss of consciousness. Death. Post-mortem showed behind extradural abscess one on floor of second left frontal convolution. Layer of healthy brain tissue between.

**HERZFELD, J.**: (1) Coryza. Left-sided frontal headaches. External operation. Posterior wall frontal sinus carious and removed. Epidural abscess found. Dura incised. Subdural abscess evacuated. Fistula in frontal lobe of brain. Recovery.

### FRONTAL LOBE ABSCESS ASSOCIATED WITH SUBDURAL.

**BERENS, T. P.**: (1) Deafness right ear. Intermittent purulent discharge. Tender mastoid. Radical operation. Sixteen days later profuse hemorrhage from middle ear cavity. Convulsions, headache, stupor, coma. Then, attack of typical Jacksonian epilepsy. Incision through dura revealed pus. Hernia of brain filling bone wound. Death some days later. Autopsy showed subdural abscess over right hemisphere. Large abscess also occupying most of right frontal lobe, with spontaneous discharge into subdural space. Author believes case to be of otitic origin.

**Goris** (second case): (1) Frontal sinusitis. Meningitis. Death three days after operation for extradural abscess. Post-mortem showed frontal lobe abscess (subdural).

**Herzfeld, J.**: (1) Coryza. Left-sided frontal headache. External operation. Posterior wall frontal sinus carious and removed. Epidural abscess found. Dura incised. Subdural abscess evacuated. Fistula in frontal lobe of brain. Recovery.

**Jessamon, L. W.**: (1) Tenderness over left frontal sinus and ethmoids. Nasal operation. Temperature. Thick speech. Aphasia. Paresis right arm. Complete paralysis right hand. Posterior wall frontal sinus removed. Congestion optic disc. Aphasia increased. Third operation subdural abscess. Death. Post-mortem showed dura thickened, especially over ethmoid and discoloration over ethmoid bone.

### FRONTAL LOBE ABSCESS ASSOCIATED WITH MENINGITIS.

**Reinhard**: (1) Purulent leptomeningitis and frontal lobe abscess.

**Manasse**: (1) Death from meningitis after abscess left frontal lobe, left temporal lobe and intrameningeal abscess in left occipital lobe.

**Zemann**: (1) Right frontal lobe abscess in form of superficial cortical abscess. Meningitis.

**Preysing**: (1) Left frontal lobe abscess following empyema of left maxillary sinus, frontal sinus, and ethmoid cells. Meningitis. Death.

**Treitel**: (1) Abscess frontal lobe. Basal surface frontal lobe hemorrhagic. Dura adherent over frontal lobe. Purulent basilar meningitis.

**von Schroeder**: (1) Right-sided retrobulbar phlegmon producing right frontal abscess. Serous meningitis right hemisphere.

**Kreckl**: (1) Left frontal abscess after frontal sinus operation, for swelling above left eye. Sinus filled with pus. Previously discharge of pus from nose and nasal polyps. Autopsy—soft meninges at base covered with thick greenish pus.

**Köhler**: (1) Abscess behind dura found after fluctuating painful swelling on left frontal tuberosity. Incised. Operation failed to arrest further extension of meningitis.

**Hanot**: (1) Abscess frontal lobe giving picture during life of tubercular meningitis.

**Weichselbaum**: (1) Abscess right upper eyelid. Eyeball intact. Pus right maxillary and frontal sinus and between right frontal sinus and dura mater. Abscess opposite in frontal lobe. Purulent pachymeningitis and leptomeningitis.

**Reiner**: (1) Abscess left frontal lobe with cerebral symptoms. Rickets and extensive caries frontal bone. Purulent meningitis at left side of base.

**Symonds, C. J.**: (1) Chr. suppuration of ethmoid sinus. Death from diffuse meningitis. Autopsy showed two abscesses under surface left frontal lobe. Dura mater beneath frontal lobe adherent to brain. Neither abscess opened lateral ventricle. Extension to orbit.

**Schaefer, H.**: (1) Orbital phlegmon w[illegible]eath from meningitis. Post-mortem showed abscess, destruction right e[illegible]labyrinth r[illegible]through lamina papyracea. resulting in osteitis [illegible]itis of o[illegible] pachymeningitis and purulent leptomeningiti[illegible]

**Uffenorde, W.**: (1) Ozena and lef[illegible]ation leading to orbital phlegmon thr[illegible]tradural abscess and frontal sinus ab[illegible] meningitis, after rupture of fronta[illegible]

**Hosch, P. H.:** (1) Bilateral maxillary sinusitis and right-sided frontal and ethmoidal sinusitis. Extradural perisinus abscess and frontal lobe abscess found at base of right frontal brain after death. Purulent meningitis.

**Heiman, Th.:** (1) Bilateral empyema antrum of Highmore and left-sided frontal sinusitis. Bilateral earache. Severe headache and vomiting. Sudden death. Autopsy showed extradural abscess. Right lobe and both frontal sinuses, ethmoid and maxillary sinus, filled with pus. Abscess with stalk. Diffuse meningitis.

**Grünwald, I.:** (1) Ozena. Empyema both frontal sinuses. Carious destruction posterior sinus wall. Right-sided pachymeningitis. Abscess frontal lobe discovered eight days after primary operation. Recovery.

**Elschnig, A.** (third case): (1) Chr. frontal sinusitis. Incipient orbital phlegmon. Radical operation after incision at upper inner orbital margin. Abscess found in frontal brain. Death. Autopsy showed abscess reaching to area of second frontal convolution. Incipient encephalitis remainder frontal lobe and adjacent temporal lobe. Purulent meningitis entire right cerebral hemisphere.

**Hirschberg, O.:** (1) Metastatic brain abscess following abscess bronchial glands. Fistula from broken down glands to esophagus. Death from purulent meningitis. Autopsy showed abscess right frontal lobe and several smaller abscesses.

**Stucky, J. A.:** (1) Acute otitis right ear. Syphilis. Metastatic abscess left frontal lobe. Very chronic as shown by firm capsule. Death from meningitis.

**Hurd, L. M.:** (1) Chr. pansinusitis. Epidural abscess; frontal lobe abscess. Meningitis. Death.

**Stucky, J. A.** (second case): (1) Traumatic ethmoiditis. Meningitis. Abscess frontal lobe. No localizing symptoms. Death. Post-mortem showed necrotic cribriform plate. Fracture through table left frontal sinus. Cerebral convolutions softened.

**Mayer, H.:** (1) Radical sinus operation for left-sided frontal sinus empyema. Death. Frontal abscess. Purulent meningitis. Thrombosis of sagittal and cavernous sinus. Bone intact.

**Roepke** (second case): (1) Bilateral frontal sinus suppuration. Osteomyelitis. Frontal lobe abscess. Meningitis. Three operations. Abscess had ruptured into meninges and into the lateral ventricle. Death.

**Goris** (second case): (1) Frontal sinusitis. Meningitis. Death three days after operation for extradural abscess. Post-mortem showed frontal lobe abscess (subdural).

**Cisneros:** (1) Frontal sinusitis. Evacuation of ethmoid. Operation for brain abscess. Disintegration large portion right frontal lobe. Death from meningitis.

**Reis, W.:** (1) Traumatic panophthalmitis. Brain abscess. Incision of eyeball; evacuation of pus. Headache, fever, coma. Death from diffuse meningitis.

**Milligan, W.:** (1) Left frontal and ethmoidal sinus disease. Supraorbital incisions. Nasal drainage. Abscess left frontal lobe; extensive basal meningitis. Death.

**Milligan, W.** (second case): (1) Left frontal and maxillary sinus disease; supraorbital incision; alveolar drainage. Abscess left frontal lobe. Extensive basal meningitis. Death.

**Reinhard:** (1) Left-sided frontal sinus suppuration and frontal lobe abscess. Extradural abscess. Meningitis and brain pressure after operation. Loss of consciousness. Death. Post-mortem showed behind extradural abscess one on floor of second left frontal convolution. Layer of healthy brain tissue between.

**Wertheim:** (1) Fever, headache, discharge from nose. Tumor over right eye. Incision voided pus. Sinus wall roughened. Death. Post-mortem showed abscess right frontal lobe. Necrosis posterior wall. Circumscribed pachymeningitis.

## Frontal Lobe Abscess.

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